A STUDY OF COLORECTAL MALIGNANCY PATTERN IN TVMCH

DISSERTATION SUBMITTED FOR
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CERTIFICATE

This is to certify that this dissertation titled **" A study of Colorectal malignancy pattern in TVMCH"** is a bonafide work of Dr.K.Rajkumar, and has been prepared by him under our guidance, in partial fulfillment of regulations of The Tamilnadu Dr. M.G.R. Medical University, for the award of M.S. degree in General Surgery during the year 2008.

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CONTENTS

INTRODUCTION

Cancers in all forms are causing about 12 percent of deaths throughout the world. Colorectal carcinoma is the one of the eight leading cancers account for about 60% of all cancer cases and deaths worldwide. Colorectal carcinoma remains a leading cause of morbidity and mortality in many developed countries. The scene is much worse in the thirld world countries like India due to absence of screening programs and basic tests like occult blood in stool, the disease is often diagnosed late in course of disease, and hence the outcome is very poor. In every year nearly 5,55,000 new cases of the colorectal carcinoma was diagnosed in developed countries. In India, the incidence of the colorectal carcinoma is low, compared to the western world. Highest incidence is observed in Bhopal and lowest in Delhi. Colorectal carcinoma is actually an eminently curable disease, provided that is detected at an appropriate stage and treated adequately. Early diagnosis is therefore the key to success with this disease.In India most of the patients present at an advanced stage due to absence of screening programs and lack of investigation facilities and because of illiteracy, especially in rural areas.

AIM OF THE STUDY

1. To study the incidence of carcinoma of large intestine admitted and treated in the Department of Surgery, Tirunelveli Medical College Hospital, Tirunelveli, according to
    1) The age of the patients.
    2) The sex of the patients.

2. To study the risk factors which predispose to colorectal carcinoma.
3. To analyse the various modes of clinical presentation of colorectal carcinoma in admitted patients
4. To study the histological and morphological types of colorectal carcinoma in admitted patients.
5. To review the methods of management of colorectal carcinoma in our hospital and outcome.

MATERIALS AND METHODS

The study includes 55 histologically proven cases of carcinoma of the large intestine (colorectal carcinoma) that were admitted in the Department of surgery, Tirunelveli Medical College Hospital, Tirunelveli, during the periods of 30 months from June 2005 to November 2007 those were admitted and treated in the various units of this hospital.

Thorough evaluation of these patients was done clinically, radiologically and other relvant investigations were done in order to arrive at a confirmatory diagnosis. Those patients who did not undergo any definitive line of managements like surgery (or) investigations were excluded from our study. Most of the patients were treated surgically. The various modalities and factors involved in aetiopathogenesis, disease presentation and treatment with regard to prognosis and morbidity profile of the patients with colorectal carcinoma were analysed.

ANATOMY OF THE LARGE INTESTINE

The large intestine extends from the ileocaecal junctions to the anus.

It is about 1.5 meter long and is divided into

- Caecum
- The Ascending colon
- The transverse colon
- The descending colon
- The sigmoid colon and
- The rectum

Caecum

Blind pouch of the large intestine projects downwards from the commencement of the ascending colon, from the ileocaecal junction.

It is usually completely covered by peritoneum.

It is 6cm long and 7.5 cm broad.

Situated in the rightiliac fossa above the lateral half of the inguinal ligaments, over the ilacus and psoas fasciae, femoral and lateral femoral cutancous nerves.

The longitudinal muscle fibres concentrated and form the three flat bands called taeniae coliae one anterior, one posteromedial and posterolateral. All three converge on the base of the appendix.

Ascending colon

About 15cm in length, extends from caecum to the right colic (hepatic) flexure.

The ascending colon lies on the iliac fascia and the anterior layer of the lumbar fascia.

Usually it is retroperitoneal, rarely it may possess an ascending mesocolon.

Taeniae coli continues from the caecum.

Transverse colon

About 45cm long, extends from the hepatic to the splenic flexures in a loop which hangs down to a variable degree between these two fixed points, anterior to coils of jejunum and ileum.

The transverse colon is completely invested in peritoneum and hangs free in the transverse mesocolon, which is attached from the inferior pole of the right kidney across the 2^{nd} part of duodenum and the pancreas to the inferior pole of the left kidney. The transverse mesocolon is attached with greater curvature of stomach by the greater omentum.

Descending colon:

Less than 30cm long, extends from spleenic flexure to the pelvic brim.

The whole of its course is plastered to the posterior abdominal wall by peritoneum, though a mesentry is present in about 20% of adult.

The descending colon lies on the lumbar fascia and the iliac fascia, ends at the pelvic brim about 5cm above the inguinal ligament.

The taeniae coli, in continuity with that of the transverse colon.

Sigmoid colon

It is usually less than 45 cm long.

Formerly known as pelvic colon, extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the S3. It is completely invested in peritoneum and hangs free on the sigmoid mesocolon.

It lies usually in the pelvic cavity, coiled in front of the rectum, lying on the peritoneal surface of the bladder (and uterus)

Rectum

It is the distal part of large intestine. Rectum is about 12cm long, extends from the sigmoid colon at the level of third piece of the sacrum, to the anal canal.

The rectum is situated in the posterior part of the lesser pelvis, in front of the lower three pieces of the sacrum and the coccyx.

The rectosigmoid junction is indicated by the lower end of the sigmoid mesocolon. The rectum ends by becoming continuous with the anal canal at the ano rectal junction.

The rectum is not straight. It is curved in an anteroposterior direction and also from side to side. The three cardinal features of the large intestines. (Sacculation, appendices epiploicae and taeniae) are absent in the rectum.

The upper part is 4cm diameter, same of the sigmoid colon, but in the lower part it is dilated to form the Rectal ampulla.

Blood Supply

As the large intestine developed from the both midgut and hindgut, are supplied by the superior mesentery and inferior mesentery artery.

The rectum is supplied by the superior rectal artery, a branch of inferior mesentery artery, middle rectal artery, arise from the anterior division of internal iliac artery, median rectal artery arising from the back of the aorta near its lower end.

The venous drainage is through the veins correspond to the arteries, thus reach the portal vein via the superior and inferior mesenteric veins.

The distal 5 to 7cm of the rectum has a dual drainage. The superior haemorrhoidal vein drains into the portal circulation by inferior mesenteric vein, the middle and inferior haemorrhoidal veins passes through the pelvic veins directly into the inferior vena cava.

Lymphatic drainage

The intramural lymphatics of the large bowel begin as a plexus beneath the lamina propria superficial to the muscularis mucosa. The lymphatics pass into submucosa, where they follow the blood capillaries. Efferent lymphatic vessels proceed radially outward through the circular and longitudinal muscle layers to communicate with an intramuscular and subserosal lymphatic plexus. Most extramural lymphatics enter the mesentry and converge towards the major arterial trunks enter the superior and inferior mesenteric nodes to the para aortic nodes.

In rectum, major portion of the lymphatic drains along the superior haemorrhoidal arterial trunk towards the inferior mesenteric artery, reaching inferior mesenteric nodes after passing through the para rectal and sigmoid nodes.

Lymphatics from the lower half of the rectum pass along the middle rectal vessels to the internal iliac nodes

Sets of lymph nodes

Epicolic nodes-lying on the wall of the gut

Paracolic nodes-medial side of the ascending and descending colon and near the mesocolic border of the transverse and sigmoid colon

Intermediate nodes-on the main branches of the vessels

Terminal nodes-on the superior and inferior mesenteric vessels

Nerve Supply

Parasympathetic supply partly from vagi and partly from pelvic splanchnic nerves.

The sympathetic supply is derived from the T10 – L2 segments.

Microscopic Anatomy

The large bowel wall is composed of six layers. Mucosa, muscularis mucosa, submucosa, mucularis propria, subserosal fat and serosa. The rectum has similar histological features but lacks of serosa.

The mucosa is the innermost layer of the large intestine contains crypts of lieberkuhin and has no villi.

The muscularis mucosae represents an external longitudinal layers. In the abdomen colon, its outer layer is thickened to form three longitudinal bands of approximately 5 to 10mm width that are termed the taeniae coli.

At the level of each taeniae is an interchange of muscle bundles bewteen circular and longitudinal layers. At the level of the distal rectal wall, the circular muscles thickens to form the valves of houston, the taeniae are replaced by anterior and posterior longitudinal musculature.

Between the circular muscle and the muscularis mucosae is the submucosa. This layer contains a vast network of blood vessels and the autonomic nervous plexes of meissner.

The muscularis propria is comprised of an inner circular layer and an outer longitudinal layer of smooth musculature.

The most external layer of the large intestine of the serosa and it represents the visceral peritoneum. All microscopic layers of the colon and rectum are significant in defining clinical and pathological tumour staging, which guides the therapeutic approaches.

EPIDEMIOLOGY

Colorectal cancer is predominanly a disease of westernized societies and environmental factors are important in its etiology.

High incidence countries are North American Countries and North Western European Countries.

Low incidence countries are sub saharan Africa, India and South America.

In USA incidence of colorectal cancer varies from 57.4 / 1,00,000 in man.

In Japan 1.33 / 1,00,000 among males.

Worldwide, colorectal carcinoma is a one among the eight leading cancers. In male, it is the third common cancer after Lung and Stomach. In female, also in third position after breast and cervix. In both sexes, it is account for fourth position after Lung, Stomach & Liver.

In India, highest incidence of colorectal cancer is observed in Bhopal (5.5 / 1,00,000). Lowest in Delhi (3.0 / 1,00,000).

According to a hospital based cancer registry report, the colorectal cancer constituted 4.7 / 1,00,000 in males 3.13 / 1,00,000 in females in India. InChennai, 3.3 / 1,00,000 in males. 1.7 / 1,00,000 in females.

Majority of the patients were in age group 45 years and above and presented as: 10% of cases – localised disease, 20% - Metastatic diseease, 70% - loco regionally advanced disease.

ETIOLOGY

Colorectal cancer may be caused (or) promoted by environmental factors, especially by dietary factors that affect the enteric milieu.

Factors involved in colorectal carcinogenesis:

- High fat and high cholesterol diet, poor fibre diet.
- Fecapetaenes.
- 3 ketosteroids.
- Pyrolysis products.
- Insufficient dietary calcium and selenium.
- Bile acids.
- Faecal PH.

High fat and cholesterol increase the bile acid secretion and an increase the incidence of the colorectal carcinoma. Bile acids increase the proliferation of gut epithelium. Cholecystectmy results in high levels of bile acids in the stool and associated with greater frequency of right sided colon cancer.

Fecapentaenes are potent mutagenie compounds found in human fecus and thought to be produced by gut microflora. Intramural level of the fecapentacnes can be lowered by fibre, vitamin C and E.

3 – ketosteroids are derived from the metabolic products of cholesterol are potential tumour promoters or initiators.

Pyrrolysis products: such as benzopyrene, produced by the broiling (or) frying of meat at high temperatures.

Calcium can reduce colorectal epithetical cell proliferation by the binding of fatty and bile acids resulting in the formation of insoluble bile salt complexes.

Vitamin D, Vitamin A and Vitamin C & E also reduce the incidence of colorectal carcinoma.

Fecal pH: Higher incidence of the colonic carcinoma is seen in subjects with a higher stool pH. The alkaline pH supports higher concentration of free bile acids and other potential carcinogens.

Clinical Risk factors

Genetic

(A) Familial polyposis syndromes

(i) Familial adenomatous polyposis (FAP) syndrome.

(ii) Gardner syndrome.

(iii) Old field syndrome.

(iv) Turcot syndrome.

(B) Hereditary nonpolyposis colorectal cancer (HNPCC) – Lynch I and II syndromes)

(C) Hereditary flat adenoma syndrome (HFAS)

Other factors

- Ulcerative colitis.
- History of previous colon cancer or poly ps.
- Irradiation of pelvis.
- Prior cholecystectomy (or) Ureterosigmoidostomy.

- Peutz – Jeghers syndrome and juvenile polyposis syndrome although heritable do not carry the increased risk of malignancy.

Familial adenomatous polyposis (FAP) Syndrome :

It is inherited as an autosomal dominant trait. The affected persons develop adenomatous polyps in the entire colon.

The polyps are not present at birth but present in late adolescence, more than a 1000 may manifest. By the fourth decade all the patients develop colonic cancer. It is also associated with ampullary adenomas.

Gardner syndrome

It is inherited as autosomal dominant trait. In addition to small and large bowel polyps, desmoid tumours of the mesentery and abdominal wall, lipomas, sebaceous cysts, osteomas and fibromas are also seen.

Old field syndrome

Consists of multiple sebaceous cysts, polyposis and adenocarcinoma.

Turcot syndrome

Is an autosomal recessive conditions associated with malignant central nervous system tumours in addition to bowel polyposis.

Hereditary Non Polyposis Colorectal Cancer (HNPCC)

Some families appear to have a high frequency of colon cancer without adenomatous polyposis of the bowel, termed hereditary nonpolyposis colorectal cancer (HNPCC). It represents 1% to 6% of colorectal cancers.

It is subdivided into

(a) Lynch I syndrome

(b) Lynch II syndrome.

Lynch I syndrome is inherited as an autosomal dominant with multiple colon cancers in the proximal colon at any age.

Lynch II syndrome also has an autosomal dominant trait with multiple colon and extra colon adenocarcinomas (familial adenocarcinomatosis) involving the ovary, pancreas, breast, bile duct, endometrium and stomach.

Amsterdam Criteria for HNPCC

(1) Histologically verified colorectal carcinoma in three or more relatives, one of whom is a first degree relative of the other two.

(2) Colorectal cancer involves at least two successive generations and

(3) atleast one family member who has developed colorectal cancer by age 50.

Hereditary flat adenoma syndrome (HFA)

Flat adenomas with diameter greater than 5mm show aneuploidy and 80% of them turn malignant.

Ulcerative colitis

For patients with Ulcerative Colitis, the incidence of malignancy increases with the extent of bowel involvement, age at onset, severity and the duration of disease.

The incidence of the colorectal carcinoma in patients with ulcerative colitis is 5.7 times higher. Patients with pancolitis for 30 years have more than 35 percent chance of developing bowel cancer.

- Previous malignant disease : Patient who have undergone treatment for a large bowel adenocarcinoma are at a three fold risk of a second colorectal tumor.
- Irradiation of the pelvis enhances the risk of sigmoid cancer.
- Previous cholecystectomy (or) ureterosigmoidostomy increases the risk of large bowel cancer.

Polyps

- Neoplastic and inflamatory polyps occur in the large bowel.
- Adenomatous polyps may be tubular (or) villious.
- Tubular adenomas are four times more common than villous adenomas.
- In general, larger polyps are more likely to contain malignant focus than the smaller ones, nearly half of polyps larger than 2cm in diameter contain malignancy.
- Villous adenomas are reported to be 8 to 10 times more likely than tubular polyps to be cancerous.

  Other polyps are,

  1. Hamartomatous polyps
  2. Inflamatary polyps

     (Ulcerative colitis – pseudopolyps)

PATHOLOGY

Site

Commest site of tumour is the rectum(38%), followed by the sigmoid colon(21%), the caecum(12%), the rectosigmoid junction(7%), the transverse colon(5.5%), the ascending colon(5%), the descending colon (4%), the splenic flexure(3%) and the hepatic flexure(2%).

Macroscopic appearance

Macroscopically carcinoma of the large intestine belongs to 4 types,

1. Proliferative (or) fungating (or) cauliflower
2. Ulcerative
3. Annular
4. Tubular

About 2/3 of the tumours are ulcerating and 1/3 are fungating.

Right sided cancers are usually proliferative.

Left sided cancer tend to grow in an annular fashion.

Microscopic Appearance

The major histologic type of large bowel cancer is adenocarcinoma, which accounts for 90% to 95% of all large bowel tumors. It is the only histologic type further classified by grade, and a number of histologic types of large bowel.

Colloid (or) mucinous adenocarcinoma represents about 17% large bowel tumors.

Rare signet – ring cell carcinoma accounts 2 to 4 of mucinous carcinomas.

Some signet – ring tumours appear to form a linitis plastica type tumour by spreading intramurally, usually not involving the mucosa.

Other rare variants of epithelial tumours include squamous cell carcinomas and adenosquamous carcinomas, sometimes called adenocanthomas.

Undifferentiated carcinomas include carcinoma simplex, medullary carcinoma, trabecular carcinoma.

Carcinoid tumour - 4% to 17% appear in the rectum. 2 – 7% may appear in colon.

Other tumors are leiomyosarcoma, (accounts for 0.1 to 0.3%), lymphoma, melanomas and unclassified tumours.

WHO classifies the malignant primary tumours as follows,

a) Epithelial tumors:

1. Adenocarcinoma.
2. Mucinous adeno carcinoma.
3. Signet ring cell adenocarcinoma.
4. Squamous cell adenocarcinoma.
5. Adenosquamous carcinoma.
6. Undifferentiated carcinoma.
7. Unclassified carcinoma.

b) Carcinoid tumors:

1. Argentaffin
2. Non argentaffin.
3. Composite.

c) Non epithetial tumors:

1. Leiomyosarcoma.
2. Others
3. Haematopoietic and lymphoid neoplasm.
4. Unclassified.

Degree of differentiation:

By the degree of differentiation, the adenocarcinoma is classifing into 3 grades.

Dukes grading systems

Grade I – Well differentiated.

Grade II – Intermediate.

Grade III – Poorly differentiated.

Clinical features

Non specific:

- Change in bowel habits.
- Intermittent abdominal pain.
- Palpable mass (common with right colon cancer)

Bleeding:

- Acute (or) as red blood mixed with stools.
- Occasionally melena in a right colon cancer.
- Chronic occult blood loss with iron deficiency anaemia and weakness.

Obstruction

- Obstruction is most commonly associated with cancer of the left colon.

- If the ileocaecal valve is competent, patient manifest as an acute abdomen due to closed loop obstruction.

- If the ileocaecal valve is incompetent, the obstruction is more insidious with increasing constipation and abdominal distension noticed over many days.

Perforation – acute (or) chronic

- Acute perforation, usually of the caecum, is clinically similar to appendicitis with pain, fever and a palbable mass.

- Chronic perforation with an internal fistula (eg – colovesical) may present with recurrent urinary tract infections (or) pneumaturia.

Other symptoms : Dyspepsia, Ball rolling movements, Jaundice, loss of appetite, loss of weight, low back pain and urinary symptoms.

Spread

(a) Local invasion:

(i) Circumferential growth.

(ii) Lateral transmural penetration.

(iii) Longitudinal spread.

(b) Lymphatic spread:

- Normal lymphatic spread through the major arteries with four sets of lymph nodes-epicolic,paracolic,intermediate and terminal lymph nodes.

- If tumours lie between two major vascular pedicles, lymphatic flow may drain in either (or) both directions.

- If the central lymph nodes are blocked by tumour, lymphatic flow can become retrograde along the marginal arcades proximally (or) distally.

In rectal carcinoma lymphatics spread to perirectal nodes followed by spreading through the lymphatics accompanying the superior haemorrhoidal vessels.

When the haemorrhoidal lymphaties are blocked, there is lateral and downward growth.

(c) Haematogenous spread:

The liver is the primary site of haematogenous metastasis, followed by the lungs.

A pulmonary metastasis can occur directly in low rectal cancers.

Rarely bone metastasis are seen in disseminated disease.

(d) Implantation :

(i) Intraluminal spread: cells from the primary tumour are shed into the lumen during manipulation and are implanted at the anastomotie sites, surgically treated haemorrhoids and fistulas.

(ii) Peritoneal seeding: Tumours infiltrating the serosa can spread transperitoneally to the pelvis. A seedling at the port of insertion sites during laproscopic colonic resection is also reported.

SURGICAL AND PATHOLOGICAL STAGING

Duke's Classification

Dukes A : Confined to the bowel wall.

Dukes B : through the bowel wall but not involving the free peritoneal surface.

Dukes C : Involvement of nodes.

Dukes D : added as modified Dukes – presence of metastases (or) advanced loco – regional disease.

TNM classification (The UICC and the AJCC Staging system)

T – Primary tumour

Tis - Carcinoma in situ:intraepithelial or invasion of lamina propria

T1 - Tumour invades the submucosa.

T2 - Tumours invades the muscularis propria

T3 - Tumour invades through the muscularis propria into the subserosa or into non peritonealised, Pericolic (or) perirectal tissues.

T4 – Tumour directly invades other organs or structures and/or perforates visceral peritoneum

N – Regional lymphnodes:

Nx - Regional lymph nodes cannot be assessed.

N0 - No regional lymph node metastasis.

N1 – Metastasis in 1 to 3 pericolic (or) perirectal lymph nodes.

N2 – Metastasis in 4(or) more pericolic (or) perirectal lymph nodes.

N3 – Metastasis in any central lymph nodes (along the course of a named vascular tree)

Metastasis

Mo – No metastasis

M1 – Metastasis

Lymphatic invasion

L0 – No lymphatic involvement.

L1 – Lymphatics involved.

Venous invasion

V0 – No vessel involvement.

V1 – Vessels involved.

Residual tumour after surgical resection

R0 – Complete tumour resection with all margins negative

R1 – Incomplete tumour resection with microscopic involvement of a margin

R2 -- Incomplete tumour resection with gross residual tumour not resected

Staging of colorectal cancer by the American joint committee on cancer

Stage 0 - Carcinoma in situ Tis N0 M0.

Stage I - Tumour invades submucosa T1 N0 M0.

Stage II - Tumour invades through muscularis propria into subserosa , or into nonperitonealized pericolic or perirectal tissues T3 N0 M0.

Tumour perforates the visceral peritoneum or directly the visceral peritoneum or directly invades other organs or structures T4 N0 M0.

Stage III - Any degree of bowel wall perforation with regional lymph node metastasis

N1 - 1 - 3 pericolic or perirectal lymph nodes involved .

N2 - 4 or more pericolic or perirectal lymph nodes involved.

N3 - Metastasis in any lymph node along a named vascular trunk.

Any T N1 M0

Any T N2 N3 M0

Stage IV - Any invasion of bowel wall with or without lymph node metastasis, but with evidance of distant metastasis.

Any T Any N M1

Astley coller (Modified Dukes Classification)

A – confined to mucosa.

B1 – invading muscularis propria.

B2 – invasion of all layers.

C1 – limited to bowel wall but with nodal involvement.

C2 – full thickness invasion through the serosa with nodal involvement.

Dukes staging system correlated with TNM

Dukes A - T1, No, M0

T2, No, M0

Dukes B – T3,No,M0

T4,No,M0

Dukes C – Any T,N,M0,AnyT, N2,M0

Dukes C2 – Any T, N3, M0

Dukes D – Any T, any N,M

Modified Astler – Coller (MAC) System

Correlated with TNM

MAC A	T,N0,M0
MAC B1	T2,N0,M0
MAC B2	T3,N0,M0
	T4,N0,M0
MAC B3	T4,N0,M0
MAC C1	T2,N1, M0
	T2,N2,M0
MAC C2	T3,N1,M0
	T3,N2,M0
	T4,N1,M0
	T4,N2,M0
MAC C3	T4, N1, M0
	T4,N2,M0

INVESTIGATIONS

Fecal occult blood test (slide guaiac test)

It plays a major role in screening study, is not sufficiently sensitive to be used as a diagnostic tool.

Rigid and flexible sigmoidoscopy

Recent studies show that only some 38% of cancers are within reach of the rigid sigmoidoscope and 60% within reach of flexible sigmoidoscope. It is used in visualise the rectosigmoid junction, which is often poorly visualised in barium enema.

Plain abdominal radiograph

In uncomplicated cases, plain X.-ray is almost always normal.Occasionally a soft tissue mass or stricture distorting the luminal gas shadow may be seen. Granular calcification in mucus secreting tumours and liver metastases may be seen.

Plain abdominal radiography has a major role in patients presenting with complication, like intestinal obstruction and perforation .

Barium enema

It is the gold standard diagnostic method of colonic carcinoma even today. In barium enema, the colonic tumours shows a large, irregular, permanent filling defect in the barium column. It is also used as therapeutic in the cases of pseudo obstruction. Barium enema is contraindication in acute intestinal obstruction and perforation.

Double contrast Barium enema

Is a remarkably accurate method of detecting primary colorectal cancer, cancer detection rate is 94-98% in the best hands (Kelvin 1982, Johnson et al 1983,Stevenson 1993) and error rate is lessthan 5%. Even a small 5 mm leison may be picked up.

Fibro optic flexible colonoscopy

It is the investigation of choice in many institutions. It is used for both diagnostic and therapeutic purposes. Main benefit of the colonoscopy is the facility to photograph and remove the polyps. Colonoscopy is also the follow up procedure of choice after resection for cancer.

Ultrasonography (Abdomen and pelvis)

It has an important role in the detection of site and extent of tumour, but it's main role is the detection of metastasis and ascitis.

Computed Tomography (CT - Scan)

CT is of value in diagnosing colorectal cancer, staging rectal tumours, detecting liver metastases and local recurrence. As a primary diagnostic test it is a useful alternative to barium enema elderly patients who are unable to retain barium and air (Day et al 1993)

MRI (Magnetic Resonance Imaging

MRI is used in preoperative assesment of spread in rectal carcinoma.

Intraoperative and laproscopic ultrasonagraphy

Autoclavable ultrasound probes are now a days used to detect the liver metastases and tumour invasion during the operative procedures as directly or through the laproscope.

Immunoscintigraphy

Radio labelled monocolonal antibodies have been used to target primary or recurrent malignant tissues.

Hydrocolonic sonography

Recently developed diagnostic method in which colon is filled with water and used as an acoustic window to visualise colon.

Endosonic Ultrasonography

Useful in the rectal cancer, the depth of the cancer invasion is studied . It shows each layers of the rectal wall separetely and more accurate than CT and MRI.

For assessment purpose

- Complete blood count
- Chest X ray
- ECG

Liver function test

- SGOT
- SGPT
- Sr. alkaline phosphatase
- LDH

Renalfunction test.

Tumour markers

- Carcino embryonic antigen (CEA)
- CEA 19 / 9
- CEA 50
- CEA 125

CEA

Screening is most valuable in preoperative evaluations. It correlates with tumour staging, recurrence and post operative survival.

Values greater than 9ng / ml (Normal 2ng / ml) is significant.

Detection of Glycoprotion antigen, TAG – 72, which is secreted by tumour cells is detected by Indium labelled Anti - TAG-72 antibodies is the recent diagnostic tool, shows 70% sensitivity. 90% specificity and 72% accuracy.

Prognostic Indicators

1. Age : Younger individuals have a poor prognosis.
2. Obstruction and perforation : Patients present with these complications have worse prognosis.
3. Tumour differrentiation : well differentiated cancer have a higher survival rate than poorly differentiated carcinoma.
4. DNA Ploidy : Older individuals with mature (diploid) DNA have a better prognosis than those with polypoid DNA.

5. Blood transfusion: Blood transfusion during surgery lower the survival rate due to immunosuppressive effect of the transfused blood and release of prostaglandin E2 from monocytes.

DIFFERENTIAL DIAGNOSIS OF CANCER OF THE COLON

Caecum and ascending colon

- ileocaecal Crohn's disease.
- appendicular abscess, mucocele appendix, inverted appendicular stump after appendicectory, appendicular mass.
- prominence of ileocaecal valve.
- right sided diverticular disease resulting in mass or abscess.
- inflammatory masses.
- ileocaecal tuberculosis, actinomycosis.
- colonic lymphoma.

Transverse colon

- Extra colonic inflamatory conditions such as acute pancreatitis, cholecystitis.
- Cancer of the stomach and pancreas invade the colon.
- Ischemic colitis at the splenic flexure.
- Localized colonic crohn's disease.

Left Colon and Rectum

- Diverticular disease.
- Crohn's disease
- Cancer in the adjacent organs, ovary, prostate and stomach.
- Endometriosis.
- Uterine masses.
- Solitary ulcer of the rectum, lipoma and cysts.

TREATMENT

The Principal treatment for the colorectal carcinoma is anatomical resection.

The enbloc surgical resection is the treatment of choice in which the resection of the diseased part, adequate amount of normal colon with removal of intermediate and central lymph nodes and ligation, division of multiple, main vascular trunks.

Surgery can be used for prevention, cure, and palliation. Surgery is the only means of cure for localised colorectal cancer. The precise operation depends on the location of the tumour. Sometimes surgery is indicated in the treatment of advance cancer. It is done mainly to relieve symptoms or complications caused by the cancer, such as obstruction and bleeding. Occasionaly, Surgery can even be curative in selected cases of metastatic cancer confined to the liver.

Preoperative preparation:

1. Full mechanical bowel preparation is essential.
2. Pre operative antibiotics.
3. Prevention of deep vein thrombosis and pulmonary embolism by prophylactic low molecular weight heparin.
4. Urinary catheterisation.

Operative treatments

a)**Carcinoma of caecum and ascending colon** :

Right hemicolectomy with ileotransverse anastomoses.

b)**Carcinoma at hepatic flexure, Proximal transverse colon**

Extended right hemicolectomy with omentectomy and ileo descending colon anastomoses.

c)**Distal transverse colon and splenicflexure:**

Extended right hemicolectmy with omentectomy (or) Left hemicolectomy and ileo sigmoid anastomoses (or) transverse sigmoid anastomoses.

d)**Descending colon**

Left hemicolectomy with transverse colorectal anastomoses.

e)**Sigmoid colon**

Left hemicolectomy (or) sigmoid resection and transverse colorectal anastomoses (or) descending colorectal anastomoses.

f)**Rectum**

Upper third : High (or) low anterior resection with colorectal anastomoses with or with out protecting loop transverse colostomy.

Middle third: Low anterior resection of rectosigmoid with colorectal anastomoses. (or)

Abdomino perineal proctosigmoidectomy with permanent end colostomy.

Lower third: Abdomino perineal proctosigmoidectomy with permanent end colostomy.

other procedures used in Rectal Carcinoma.

- Abdomino Sacral resection of rectum.
- Hartmann Resection.

- Pull through procedure.
- Colo anal anastomoses.

Chemotherapy

The three major forms are :

1. Adjuvant chemotherapy for curatively resected high risk stage II and stage III colorectal carcinoma
2. Palliative chemotherapy for advanced colorectal cancer.
3. Non - adjuvant chemotherapy for non - resectable liver - only metastases.

5 - Flurouracil (5- FU) is the only drug which is very effective in colorectal carcinoma and is used world wide.

The standard regime is 5 - FU in the dosage of 500 mg/m2 daily for five days, 4 - 5 weeks. Folinic acid is added to reduce the adverse effects of the 5 - FU.

Chemotheropy with 5-FU combined with levamisole (an antihelmintic immuno modulatory drug) reduce the recurrence rate significantly.

The recent introduction of two novel agents, Irinotecan and oxaliplatin, has led to the development of new and highly active regimens. Treatment with Irinotecan added to 5 FU (FOLFIRI regimen) as a first-line therapy for metastatic colorectal carcinoma has produced superior response rate.

Radiotherapy:

Radiotherapy as a single modality of treatment has the advantage of being able to provide local palliation without systemic side effects. Symptomatic bone and brain metastases are two examples. Preoperative (or) Postoperative radiotherapy can reduce the recurrence rate.

Chemoradiation:

5-FU is a known radiosensitising agent and is sometimes given concurrently with radiation therapy. Postoperative radiation therapy and chemotherapy given concurrently have become the standard of care for stages II and III rectal cancer. This multimodality treatment improves local control and disease free and over all survival.

Chemoradiation is also used to treat patients with unresected locally advanced rectal cancer.

Pre - operative chemoradiation aims to downstage the primary cancer sufficiently for sphincter - sparing operation to be carried out. This approach will help some of the patients to avoid an abdomino perineal resection and colostomy.

Chemoprevention

Cohort studies suggest that calcium and folate supplementation may prevent the development of colorectal cancer.

NSAID - s and cyclo - Oxygenase II (COX - 2) inhibitors like celecoxib have been shown to reduce the incidence of polyps in familial adenomatous polyposis. The

US Food and Drug Administration has approved the use of celecoxib for the chemoprevention of polyps in FAP.

Specific managements problems in colon Cancer

Synchronous cancers

Synchronous colorectal cancers occur in 3% to 5% of patients.

Preoperative examination of the remaining colon in recommended either by air contrast barium enema (or) colonoscopy.

Obstructing Cancers

Obstructions are usually found in Left sided cancers.

Left sided colon obstruction will be managed by

(a) Three stage operative approach

(1) Diverting transverse colostomy.

(2) Tumour resection after 10-14 days.

(3) Closure of colostomy.

(b) Two stage – Hartmann procedure

The tumour is resected, the proximal colon brought to the skin as an end colostomy, the distal colon is closed by sutures.

Obstructing cancer in right side are usually treated with a single stage resection and ileo colic anastomoses.

Perforating cancers:

An early diagnosis and intervention with surgical resection, colostomy, peritoneal cavity irrigation, drainage and antibiotics administration are necessary to salvage the patients.

Contiguous organ involvement:

Direct involvement of the adjacent organs needed extended surgery.

Cancer in Polyps:

Cancer is present in about 5 percent of the adenomatous polyps.

Polypectomy alone is sufficient in almost all patients with a moderately (or) well differentiated cancer and with no histopathological evidence of a lymphaic vessel invasion.

Large benign sessile adenomas may require surgical resection.

Treatment of metastatic carcinoma:

Liver metastases

- Anatomically resectable with enough liver function – hepatic resection.
- Diffuse metastases – systemic chemotherapy, portal arterial infusion chemotherapy.

Extra hepatic metastases:

Loco regional recurence - complete resection.

Pulmonary metastases – pneumonectomy (or) lobectomy.

RECENT ADVANCES IN TREATMENT

Adjuvant immunotherapy in the form of tumour targeting monoclonal antibodies and autologous tumour vaccines.

Antibodies to Co - 17 - 1A, TAG 72, CA-19-9, CEA, L6 and 28A 32 are on trial.

CETUXIMAB, an epidermal growth factor inhibitor (EGFR), BEVACIZUMAB, an angiogenesis inhibitor are the newer agents which combined with IRINOTECAN may be beneficial in the untreated advanced colorectal carcinoma.

- Laproscopic colorectal surgery
- Intra luminal shield devices
- Laser lumenisation and endoluminal stenting in the management of obstructing rectal or low colonic tumour.
- Vaccination : Modified autologous tumour cells along with BCG is on trial.

FOLLOW-UP AFTER POTENTIALLY CURATIVE SURGERY

- History and physical examination and fecal occult blood every three months then every 6 months for 3 years.
- Colonoscopy, 6 months after surgery and later once a year for 3 years.
- Alkaline phosphatase every 3 months for 3 years. CEA, every 3 month for 3 years followed by every 6 months for 2 years.
- Sigmoidoscopy for rectal cancer, every 6 months for 5 years.
- Chest X-rays yearly for 5 years.
- USG / CT Scan if symptamatc or investigation are abnormal.

NONADENOCARCINOMA MALIGNANCIES OF THE COLON

CARCINOID

Carcinoid is found throughout the gastrointestinal tract.

65% present in the appendix, jejunum, and ileum.

The rectum is the next most frequent site, accounts for 18%

Other gastrointetinal tract sites, including the duodenum, stomach, colon and meckel's diverticulum, accounts for approximately 10% of carcinoid tumour

Malignant carcinoid syndrome of colonic origin occur due to liver metastases, because of the draining of serotonin from the gastrointestinal tract to the liver.

Diagnosed by colonoscopy, and serum serotonin, urinary 5-hydroxy indole acetic acid.

Primary resection is the treatment of choice.

Liver metastases – curative resection

Non – resectable – palliative debulking

Hepatic arterial embolization and chemotherapy with streptozotocin and 5 FU may be beneficial

Sarcoma

Sarcomas of the colon are rare and account for less than 0.1% of all colorectal malignancies.

Most common type is leiomyosarcoma. Other types such as fibrosarcoma and angiosarcoma are rare. A low grade desmoid tumour may involve the large bowel. Kaposi's sarcoma have been reported recently, associated with HIV infection.

Wide resection of the colon with omentectomy is the treatment of choice.

Lymphoma

Primary lymphoma of the colon accounts for 0.5% of colonic malignancies. 70% of lymphomas found in the caecum. The next most common sites are the rectum and ascending colon. The majority of the colon lymphomas are single (86%). Histologically lymphomas of the colon are similar to that other parts of the gastrointestinal tract. Most of the them are non-Hodgkin's lymphoma.

Treatment in resection of the tumour with mesentry, chemotherapy and radiotherapy are recommended.

RESULTS AND DISCUSSION

I. INCIDENCE OF COLORECTAL CARCINOMA

During the period of 30 months from june 2005 to November 2007 in the Department of Surgery, Tirunelveli Medical College Hospital, Tirunelveli, about 16273 patients were admitted. Out of those, 55 patients were histopathologically proved cases of colorectal carcinoma.

The incidence of colorectal carcinoma in TVMCH during the prescribed periods

= 55/16273 = 0.33%

II. AGE INCIDENCE

In our study of fifty five patients, the youngest one was 25 years old male patient and oldest one was eighty years old.

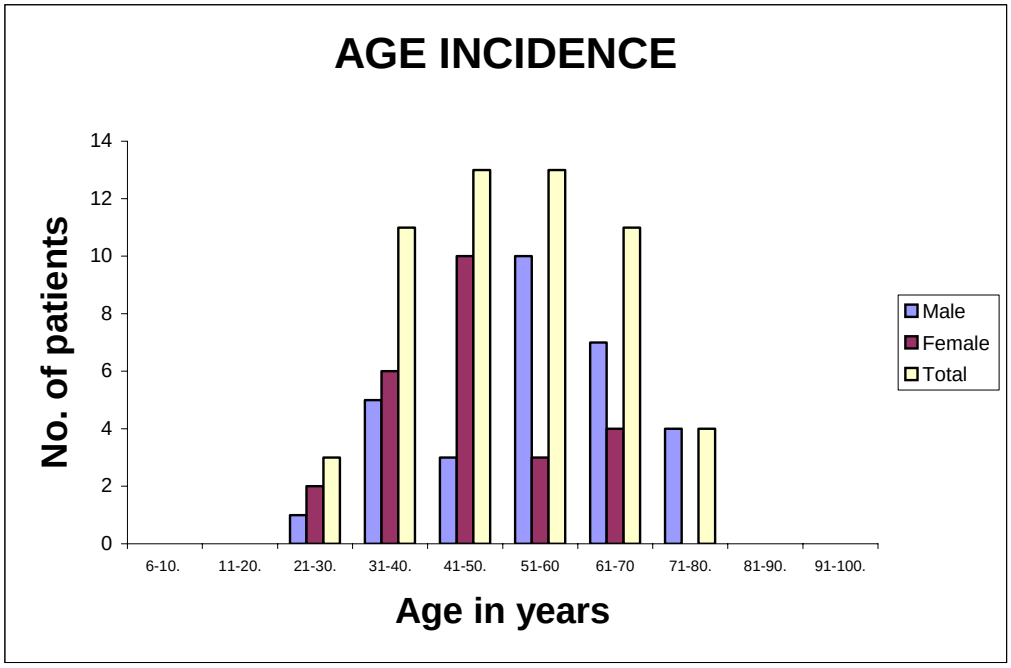
Irrespective of pathology the maximum incidence was occured between the age of 41-50 & 51 – 60 years.

Age in years	Incidence		Total	Percentage %
	Male	Female		
6-10	0	0	0	0
11-20	0	0	0	0
21-30	1	2	3	5.45
31-40	5	6	11	20
41-50	3	10	13	23.63
51-60	10	3	13	23.63
61-70	7	4	11	20
71-80	4	0	4	7.27
81-90	0	0	0	0
91-100	0	0	0	0

In male, peak incidence was between 51 – 60 years of age.

In female peak incidence was 41 – 50 years.

The reported study shows the peak incidence between 41-50 & 51 – 60 years of age.

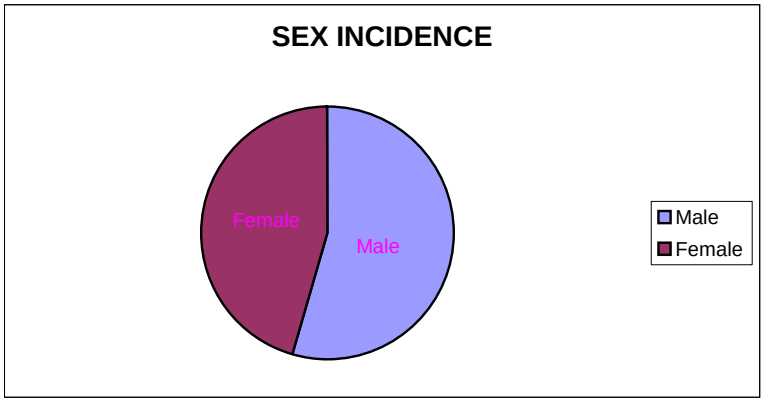


III. SEX INCIDENCE

Colorectal carcinoma is more common in male than women. In our series, out of 55 cases male to female ratio is 1.2:1, being 30 males and 25 females. The slight high incidence among the male is probably due to increased association with high fat diet, smoking and alcohol consumption compared to females.

Hospital based cancer registry, Trivandrum shows, male and female ratio was 1.5:1.

Sex	No. of Cases	Percentage %
Male	30	54.54
Female	25	45.46



IV. SOCIOECONOMIC STATUS

Out of fifty fout patients studied, the most of the patients belongs to the low socio-economic status. The socio-economic status palys an important role in prognosis of the disease. The poor socio-economic status attributed the lack of medical facilities, illiteracy, lack of awareness, poor nutrition which causing impaired immune response and wound healing.

V. PERSONAL HABITS AND RISK FACTORS

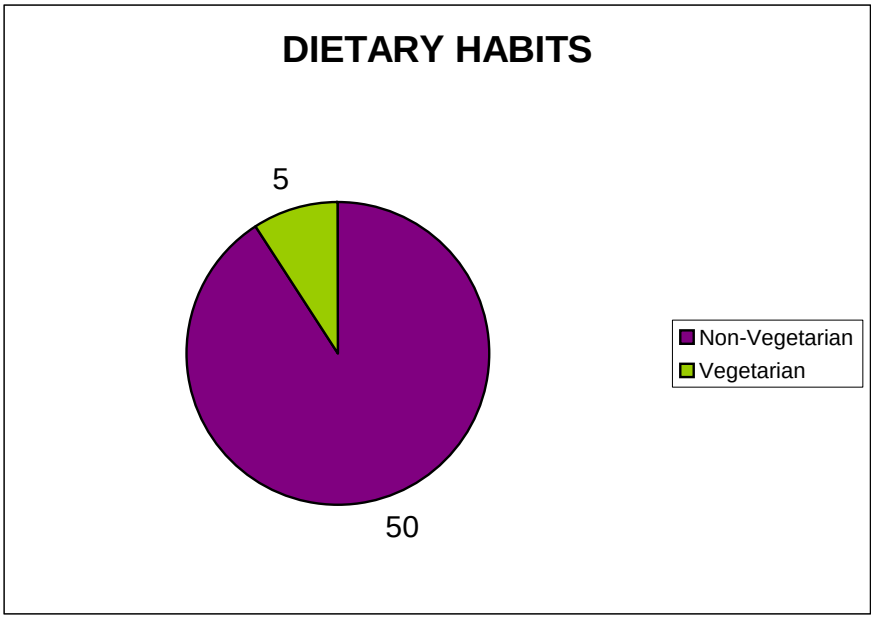
Diet plays an important role in the pathogenesis of colorectal carcinoma. In our study most of the patients were non-vegetarians. (50 persons out of 55).

The most of the male patients were smoker and alcoholic. (22 persons were smoker, 15 persons were alcoholic).

In the recent studies, the smoking increase the incidence of colorectal carcinoma (National Cancer Centre, Singapore, Cancer Update, Volume – I, 2004)

	No. of Cases	**Percentage %**
Non-Vegetarian	50	90.9
Vegetarian	05	9.1
Smoker	22	40
Alcoholic	15	27.27

Willett et al 1990 demonstrated those taking the most fat (over 65g/day) were at 1.9 times greater risk than those taking the least fat.



VI. CLINICAL PRESENTATION OF THE COLORECTAL CARCINOMA

(a) Symptoms of the colorectal carcinoma

The most common clinical symptoms are

Bleeding per rectum.

Pain abdomen

Changes in bowel habits

Abdominal Swelling

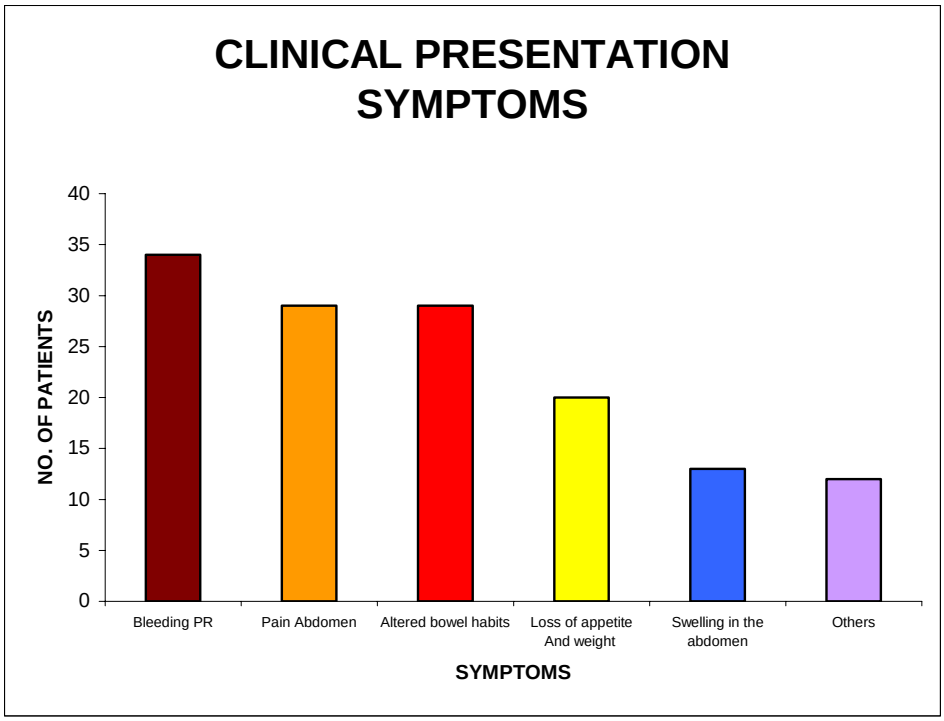
Loss of weight

Loss of appetite

Others

In our study apart from the acute cases, bleeding per rectum is seen in 34 patients, pain abdomen in 29 patients, altered bowel habits in 29 patients abdominal swelling in 13 patients loss of appetite and weight in 20 patients and others (12 nos).

Sl.No.	Symptom	No. of Cases	Percentage %	Reported Study Dent et al 2000
1.	Bleeding PR	34	61.81	60-90%
2.	Pain Abdomen	29	52.7	50-70%
3.	Altered bowel habits	29	52.7	50-70%
4.	Loss of appetite And weight	20	36.36	Above 40%
5.	Swelling in the abdomen	13	23.63	30-40%
6	Others	12	21.81	20%



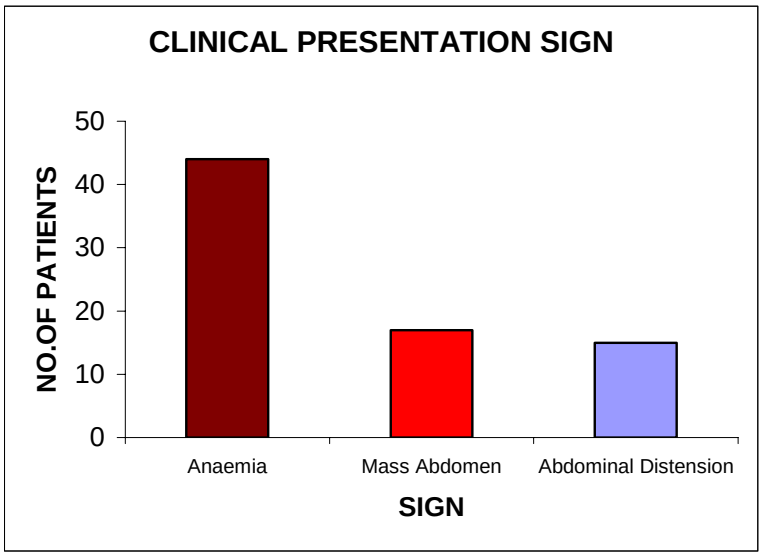
b. Signs of the colorectal carcinoma

The most important signs of the colorectal carcinoma are anaemia, mass abdomen, abdominal distension and ascities.

In our study most of the patients were anaemic (44 patients).

Abdominal mass was palpated in 15 patients including hepatomegaly in 2 person, abdominal distension was present in 12 patients including ascities in 2 patients,

Signs	No. of Cases	Percentage %
Anaemia	44	80
Mass abdomen	17	31
Abdominal Distension	15	27.27

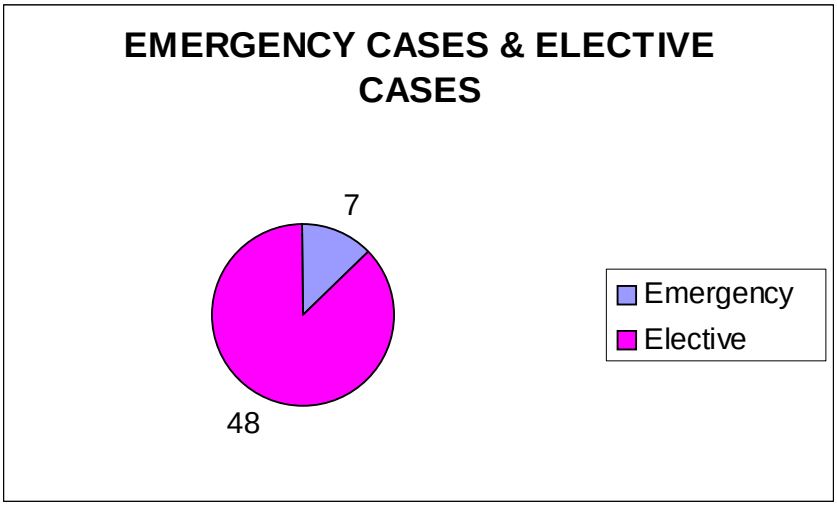


c. Patients admitted with emergency

Out of 55 patients, 7 patients were admitted as an acute emergency.

In those 7 patients, 6 patients were diagnosed as an acute intestinal obstruction and one was a sub acute intestinal obstruction.

Percentage of the intestinal obstruction with colonic carcinoma is 12.72%



(Phillips et al 1985 – obstruction occurred in 16% of the patients with colorectal carcinoma.

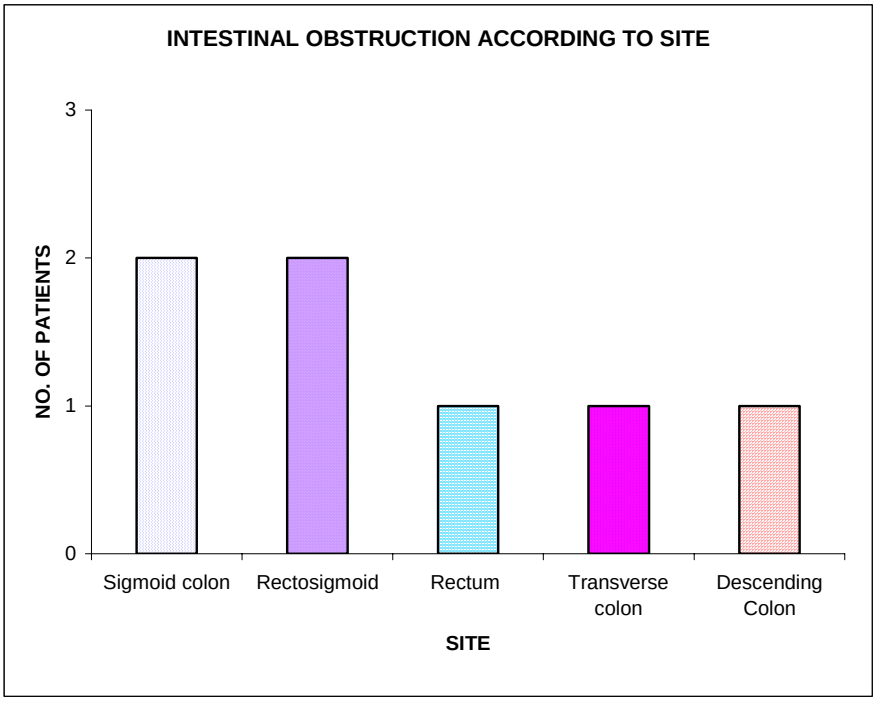
Dean et al 1994 – 8- 29% of colorectal carcinoma obstructed).

Out of 7 intestinal obstruction patients one had intestinal perforation with fecal peritonitis.

Intestinal obstruction according to the site of the tumour are as follows:

Site	No. of cases	Percentage %
Sigmoid Colon	2	28.57
Rectosigmoid	2	28.57
Rectum	1	14.28
Transverse colon	1	14.28
Descending Colon	1	14.28

Reported study shows, obstruction due to the carcinoma of the large intestine is common in left sided cancer.

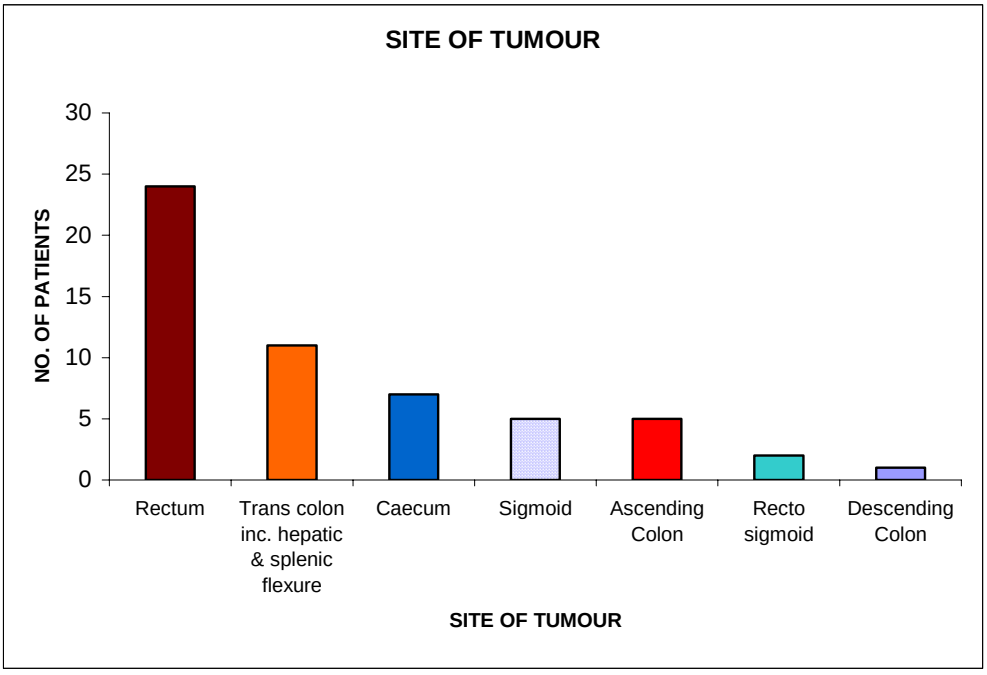


VII. PATHOLOGY

a. Site of the tumour

In our study, the most common site of the tumour is the rectum (24 patients), followed by the transverse colon including hepatic & splenic flexure(11 patients),the caecum (7 patients), the ascending colon & sigmoid (each 5 patients), the rectosigmoid (2 patients)and the descending colon (1 patient).

Site of Tumour	No. of cases	Percentage %	Reported Study Falter et al
Rectum	24	43.63	38
Trans colon inc hepatic & splenic flexure	11	20	21
Caecum	7	12.72	12.5
Sigmoid	5	9	10.5
Ascending Colon	5	9	5
Recto sigmoid	2	3.6	4
Descending colon	1	1.8	2.1



b. Macroscopic studies

Macroscopically colorectal carcinoma belongs to 4 types.

1) Ulcerative
2) Annular
3) Tubular
4) Cauliflower

The reported study shows the most of the tumour belongs to the ulcerative type (2/3 of the total).

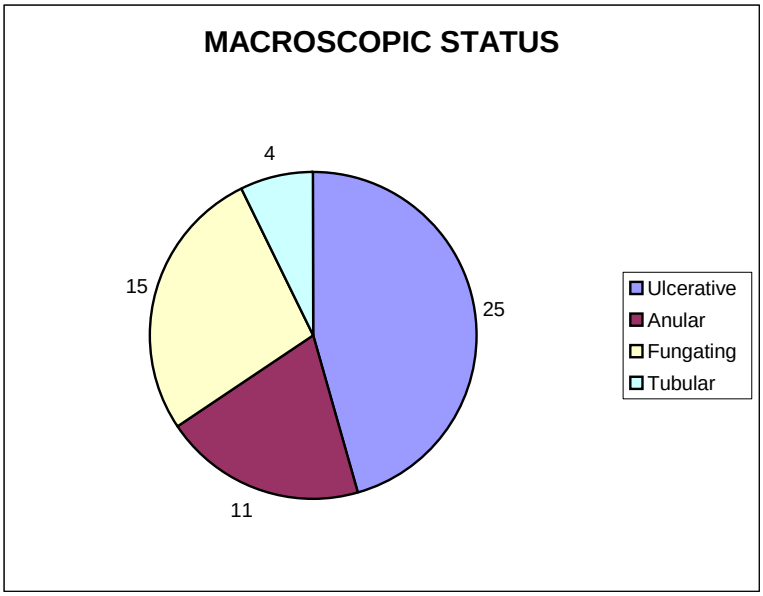
In our study 25 were the ulcerative types, 11 were annular, 15 were cauliflower(fungating),4 were tubular.

c. Microscopic studies

The major histological types of colorectal carcinoma is adenocarcinoma.

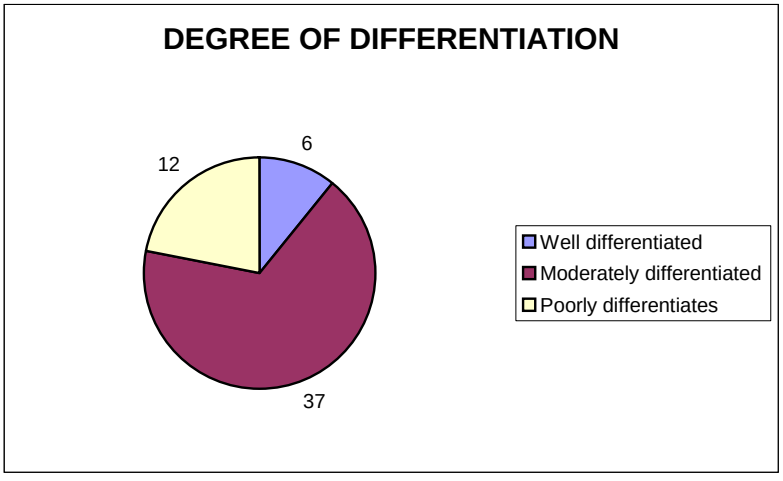
In our series of 55 cases all cases were proved as an adenocarcinoma in various differentiations.Out of 55 adenocarcinoma 37 was moderately differentiated adenocarcinoma, 6were well differentiated adenocarcinoma and 12 were poorly differentiated adenocarcinoma.

Histopathological Type	No. of Case	Percentage %
Adenocarcinoma	**55**	**100**



Differentiation Degree	No.	Percentage %	Broder's Study
Well differentiated	6	10.9	20
Moderately differentiated	37	67.27	60
Poorly differentiated	12	21.81	20

A study of Morson 1967, shows almost all colorectal malinancies are adenocarcinoma.

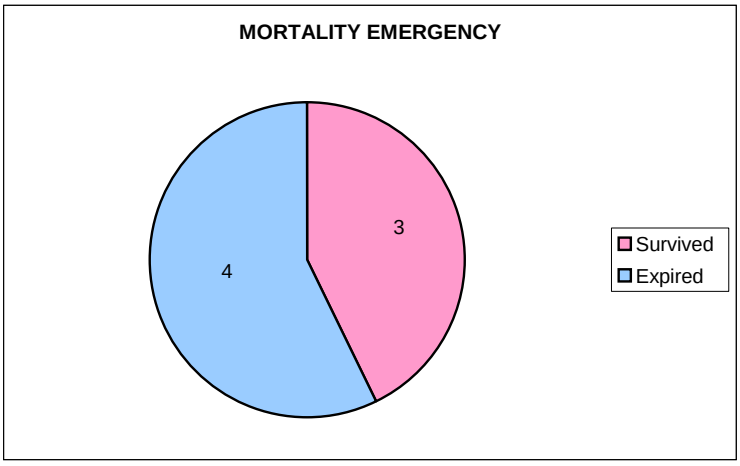


VIII. MANAGEMENT

In our study all the patients, were treated surgically by the emergency or elective surgical procedures.

Emergency Surgical Procedures

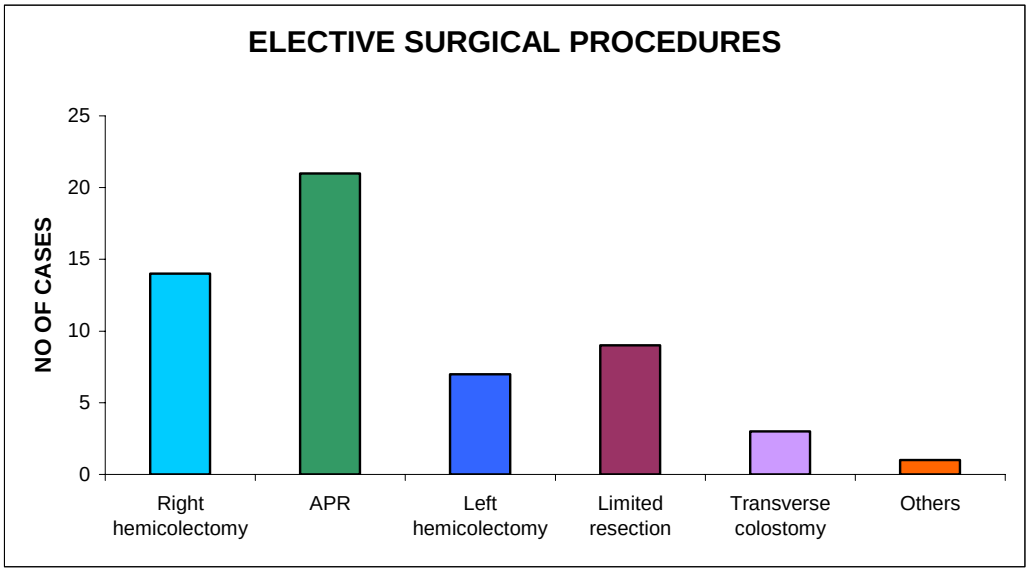
7 out of 55 patients were treated by the emergency procedures, as the patients had been admitted with acute intestinal obstruction. Three of the surgical procedures were palliative such as defunctioning colostomy and others were curative resections.



Elective Surgical Procedures

In our series, curative surgical procedures like right hemocolectomy, left hemicolectomy, extended right and left hemicolectomy, anterior resection, abdomino perineal resection and various anastomoses were done for 47 patients.

Palliative surgical procedures were done for 4 patients and biopsy was taken for 1 patients.



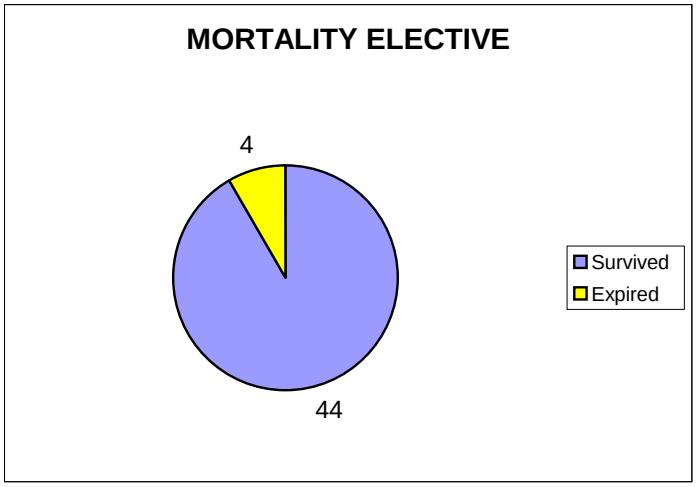
IX MORTALITY

a. Following the emergency surgical procedures

Out of 7 emergency surgery, 4 patients expired on the post operative period due to various post operative complications, 2 persons had undergone further elective curative procedures.

b. Following the elective surgical procedures

Four patients were died post operatively due to various causes like pulmonary embolism, myocardial infarction etc.,



X. FOLLOW UP

Post operative followup is aimed at detection of reccurrent tumours and re-assurance of the patient. As 80% of recurrence become apparent within years of surgery, follow up should be intense during this periods.

In our series, all patients were advised to come for regular follow up. 35 patients came regularly and completed the course of chemotherapy. One patient is refered to higher centre for further managements. Five patients had an incomplete chemotherapy. One patient who went for abdominal perineal resection readmitted with liver secondaries and ascities and died on fifth day.

During the period of follow up detailed enquiry in to the symptamatology and careful clinical examination for local recurrence and distanct metastases were made. If there is any suspicion , the cases were evaluated by USG, chest x-ray, barium enema, CT scan and colonoscopy. Though all patients were followed up in immediate post oeprative period, subsequent follow up was poor in our series. The period of follow up range from 1 month to 2 years and average period of follow up was 1 year only.

CONCLUSION

This study involves a very small subset of patients with colorectal carcinoma. The peak incidence of colorectal carcinoma in our region is in fifth decade. According to the western study reports the peak incidence is in the seventh decade.

Our study revealed a male preponderance with a male to female ratio of 1.2:1. High intake of fatty diet, smoking and alcoholism among the male population increase the risk of colorectal carcinoma in our region.

In our study, the most of the patients were diagnosed in the late stage of the disease. Commonest clinical symptom was bleeding per rectum which was correlated with other studies. The commonest sign was anaemia. A palpable mass was present in 31% of our patients. 7 patients (12.72%) were admitted as an acute emergency with intestinal obstruction. The most common site of the tumour which produced intestinal obstruction was sigmoid colon and rectosigmoid (57.14%).

The common macroscopic type of carcinoma was ulcerative type and the microscopic type was moderately differentiated adenocarcinoma. Emergency surgery was done in 7 patients, 4 patients were expired post operatively. So the mortality rate in emergency surgery was high.

Some of our patients have to travel long distances for chemotherapy and radiotherapy and end up subsequently as defaulters of these adjuvant modalities. So surgery is often the only treatment given. Hence early diagnosis and surgical resection are the best options for better results in our region.

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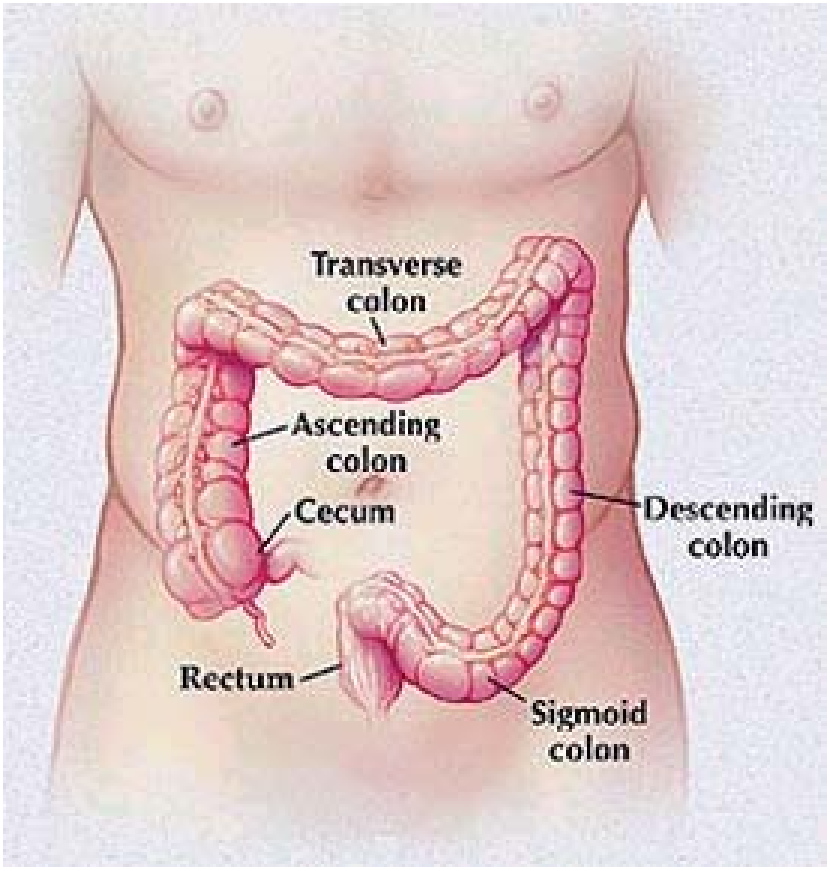
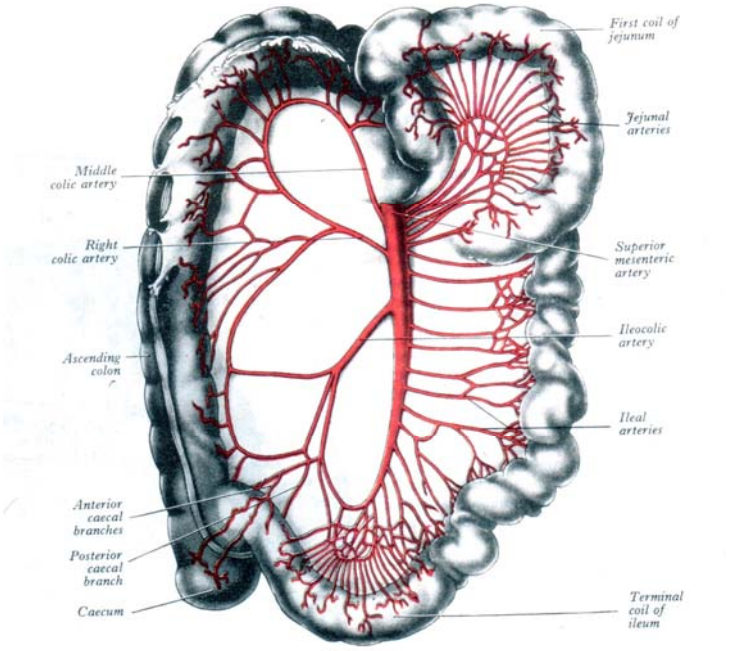
ANATOMY OF THE LARGE INTESTINE



ARTERIAL SUPPLY OF LARGE INTESTINE



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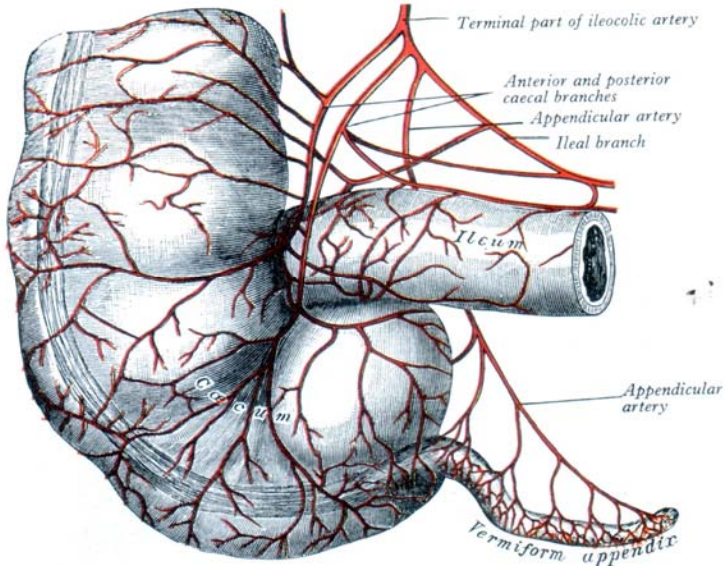
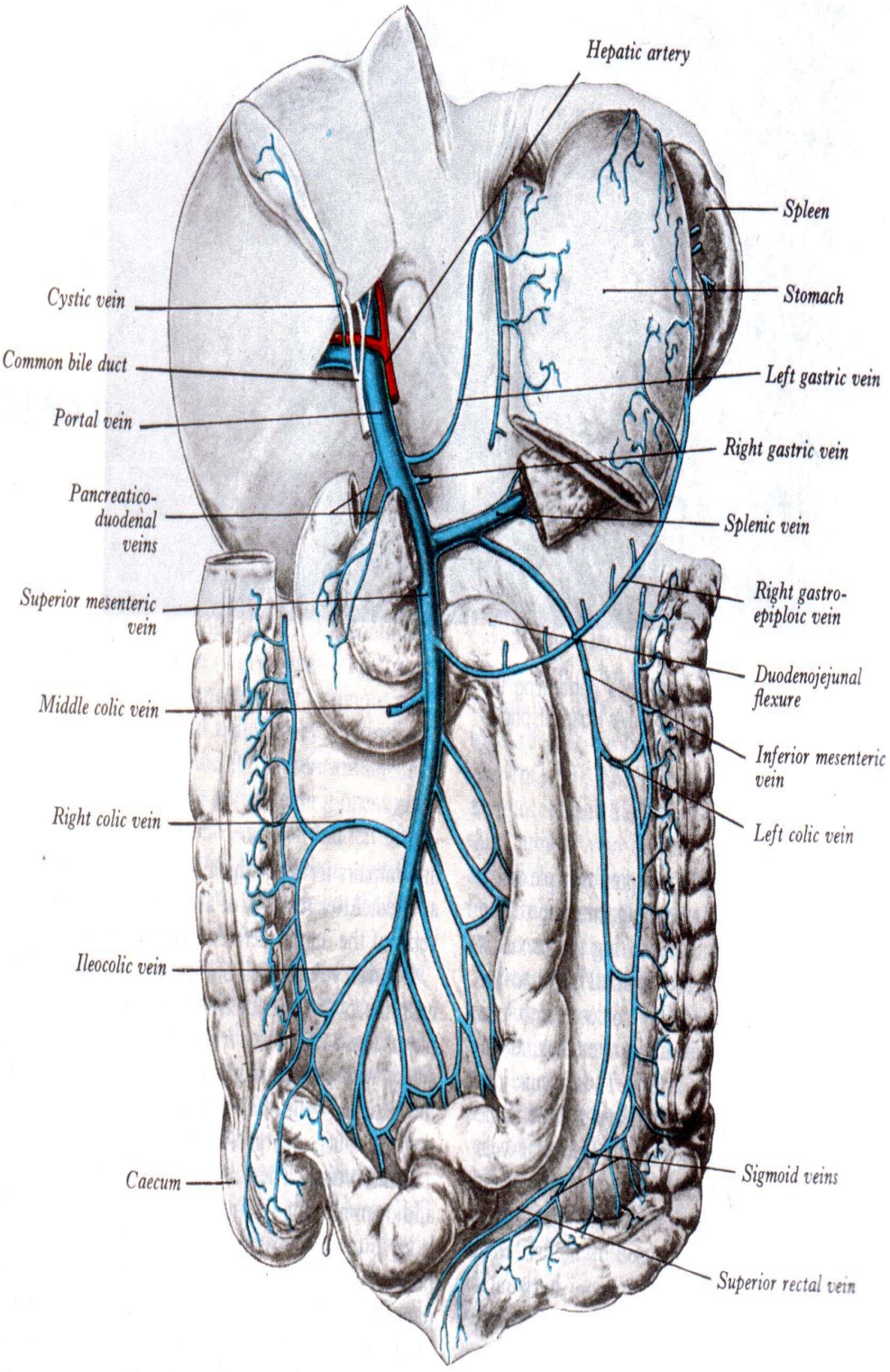
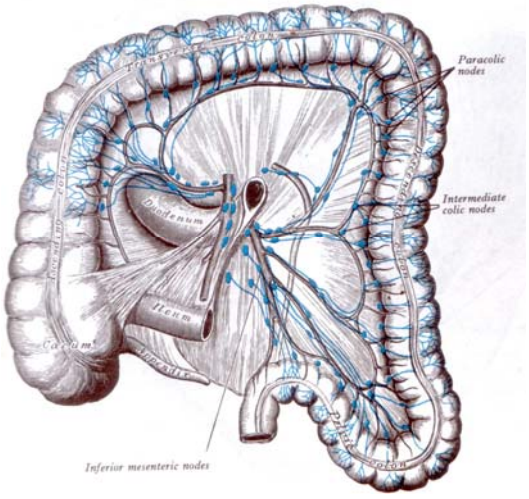


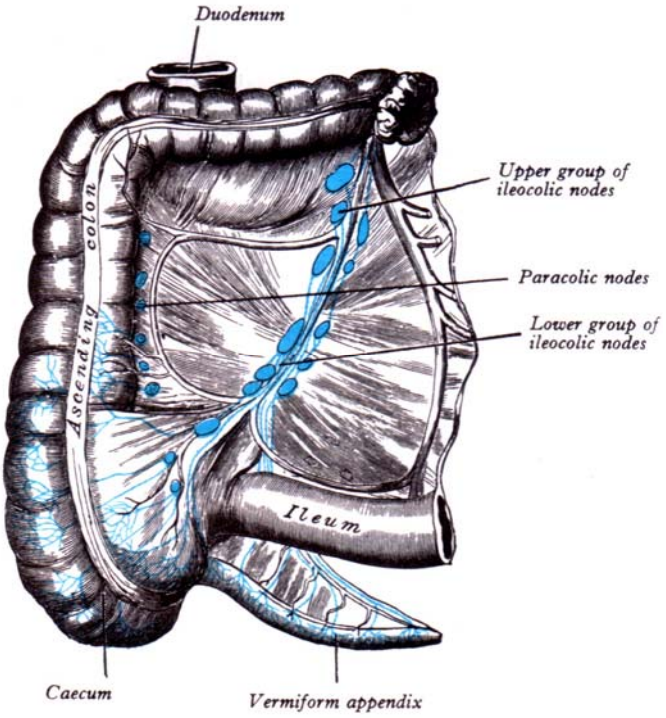
VENOUS DRAINAGE OF LARGE INTESTINE



LYMPHATIC DRAINAGE OF LARGE INTESTINE



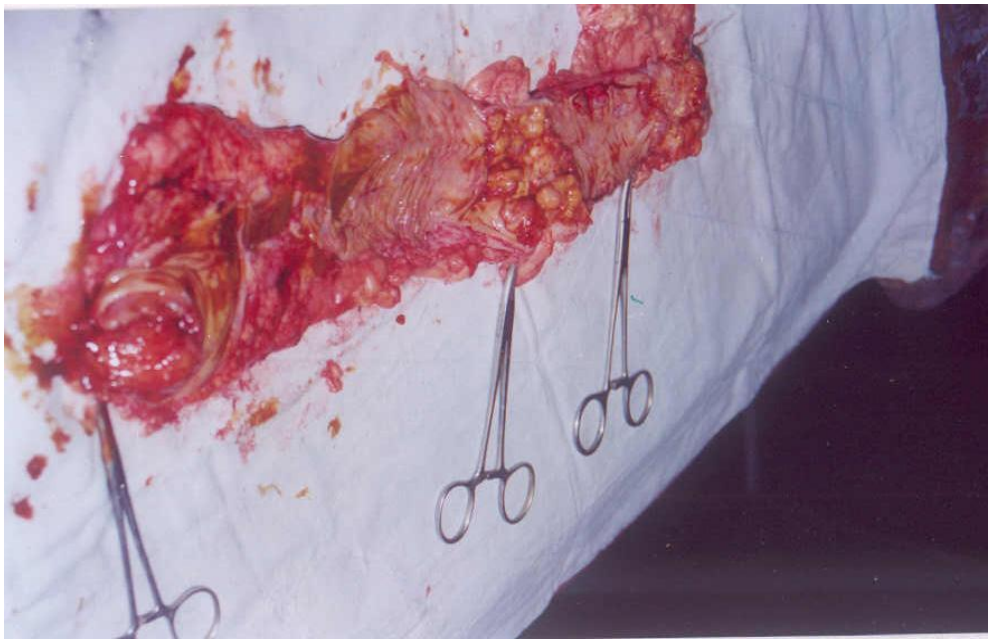
LYMPHATIC DRAINAGE OF CAECUM



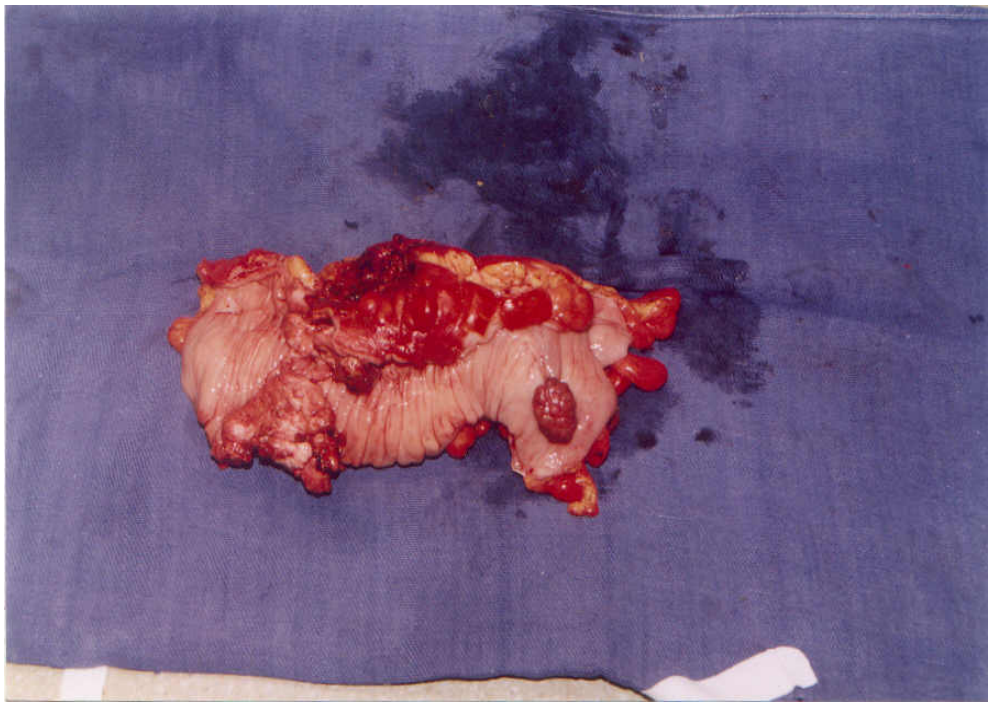
TRANSVERSE COLOSTOMY

PATIENT WITH INTESTINAL OBSTRUCTION DUE TO COLONIC GROWTH

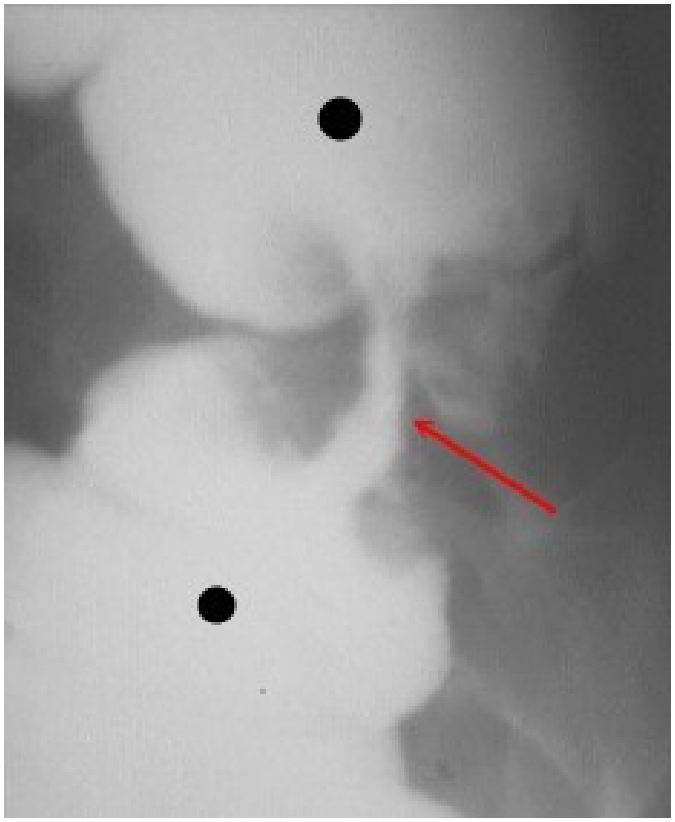
SYNCHRONOUS COLORECTAL CARCINOMA INVOLVING CAECUM ASCENDING COLON AND TRANSVERSE COLON



MULTIPLE ADENOMATOUS POLYPS

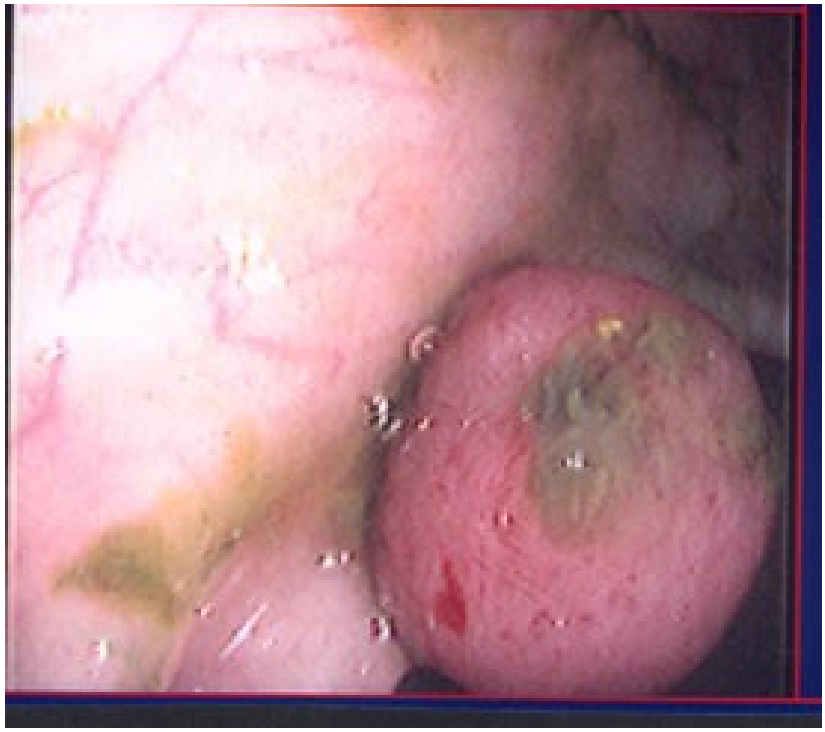


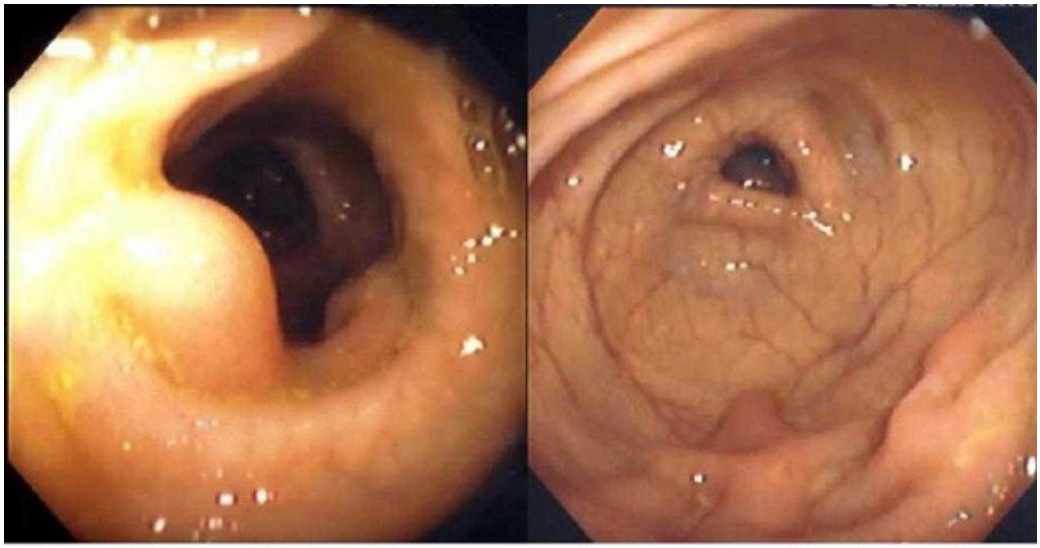
BARIUM ENEMA PICTURE SHOWS GROWTH SIGMOID COLON



DOUBLE CONTRAST BARIUM ENEMA STUDY SHOWS SIGMOID GROWTH

COLONOSCOPY VIEW OF COLONIC GROWTH

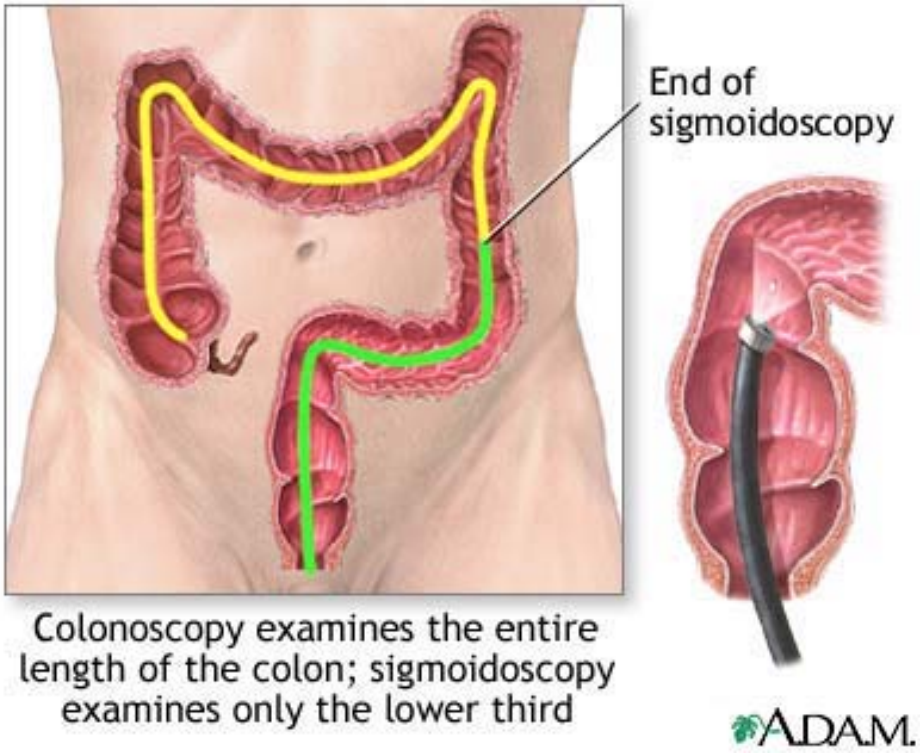




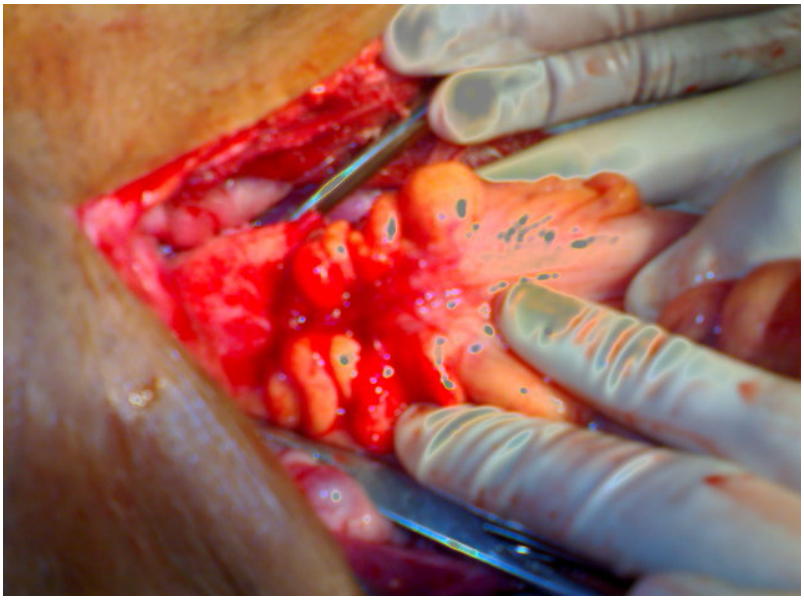
Prominent, irregular, erythematous fold in the transverse colon due to colon cancer.

BARIUM ENEMA PICTURE - CAECAL GROWTH

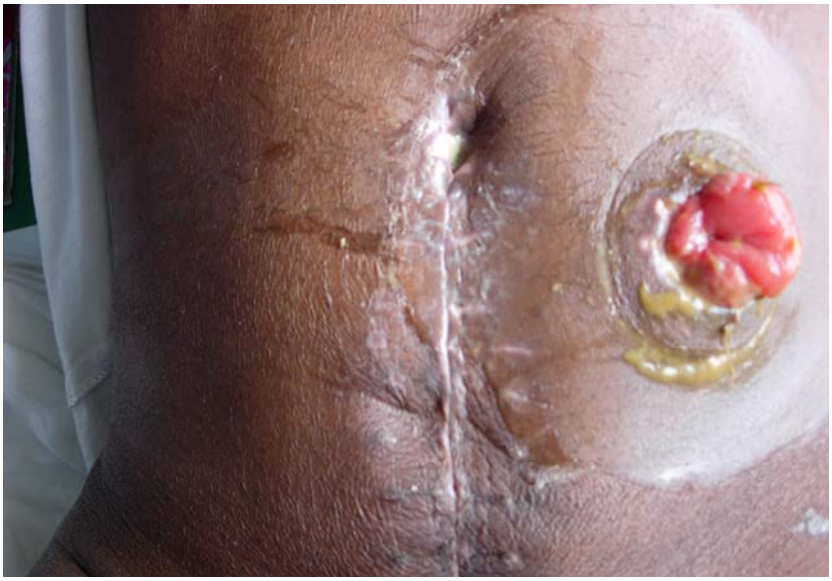
SIGMOIDOSCOPY & COLONOSCOPY



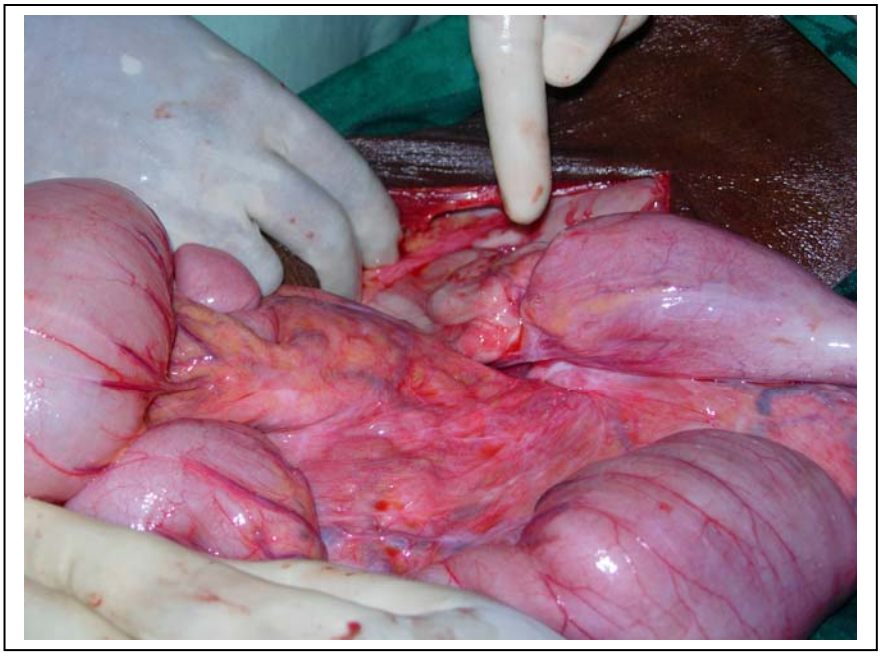
CARCINOMA RECTUM



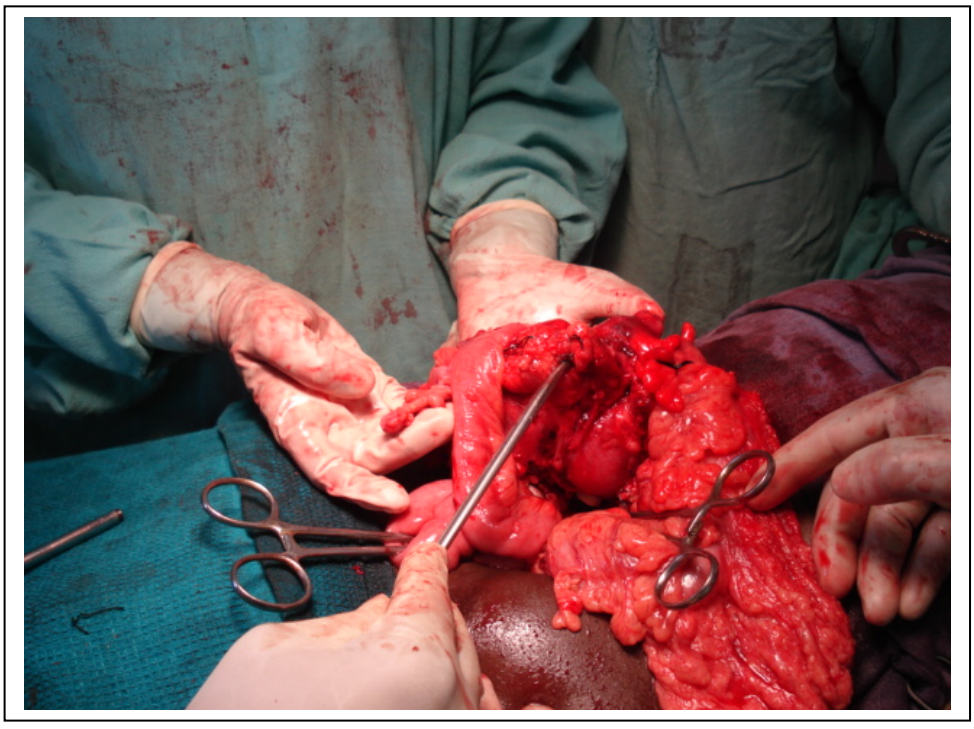
COLOSTOMY FOLLOWING APR



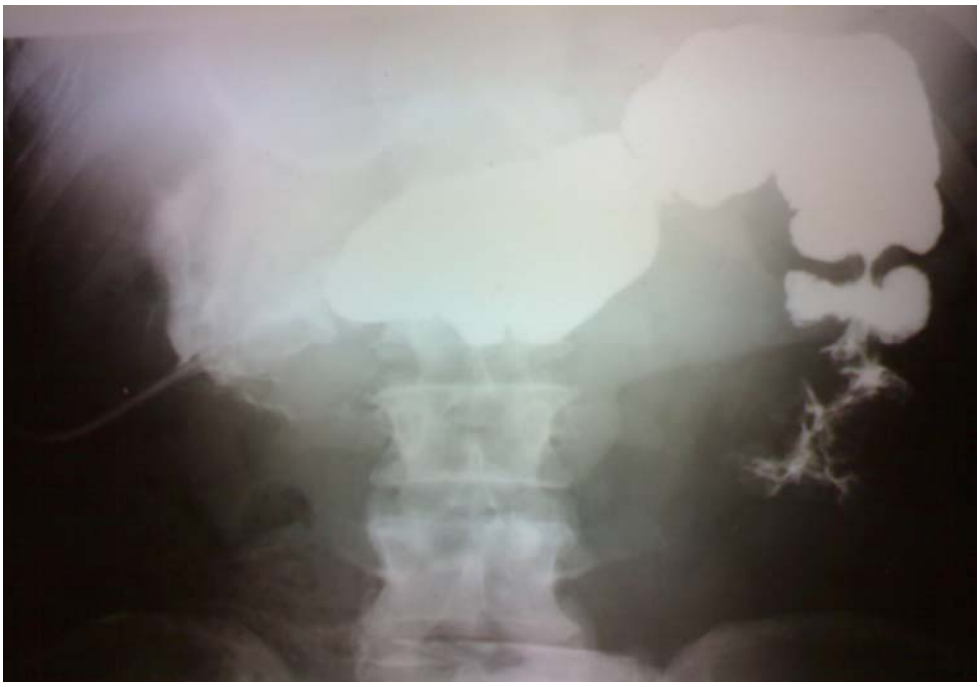
CARCINOMA CAECUM



RIGHT HEMICOLECTOMY IN PROGRESS

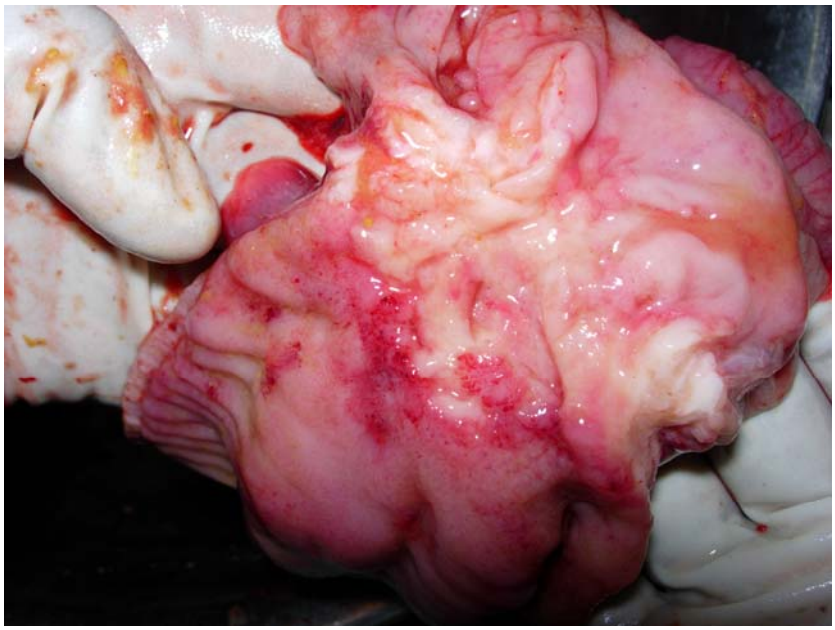


BARIUM ENEMA SHOWING GROWTH DESCENDING COLON

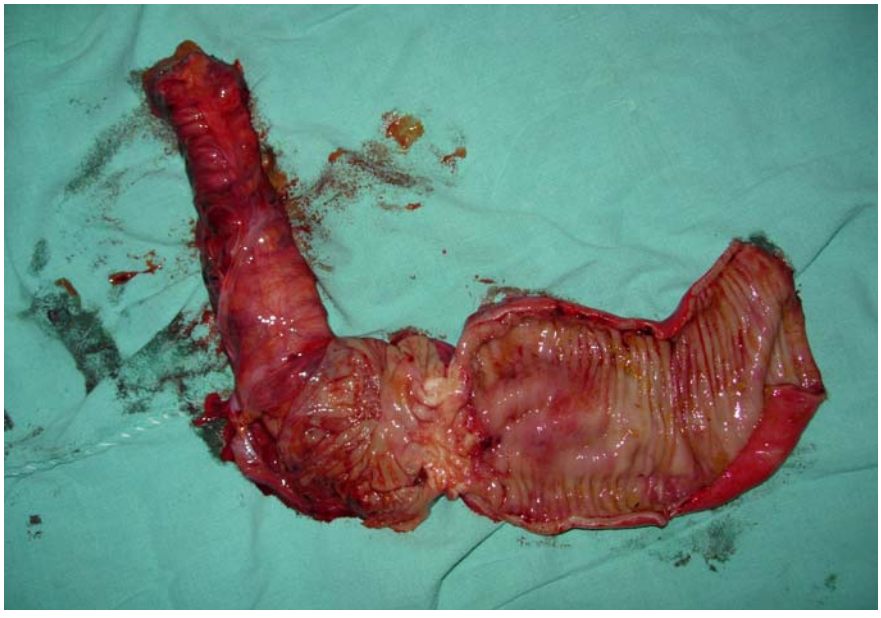


BARIUM ENEMA SHOWING GROWTH SPLENIC FLEXURE

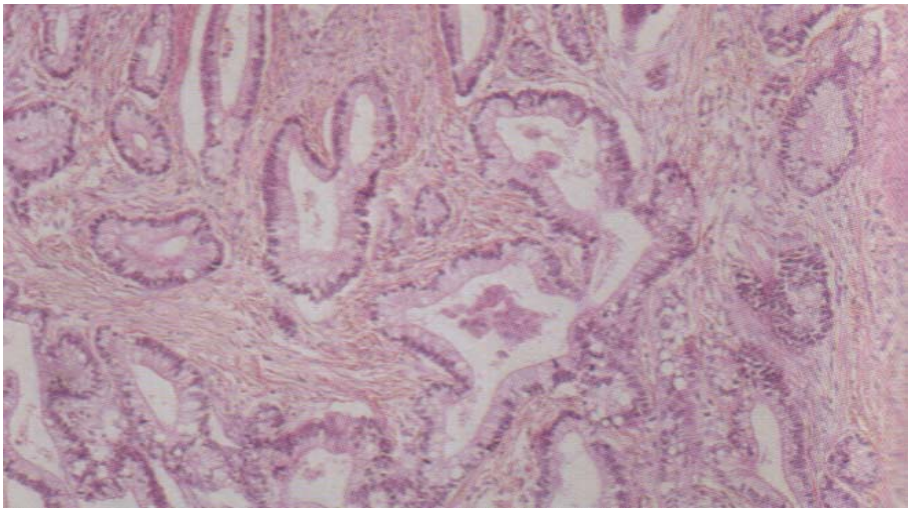
AFTER RIGHT HEMICOLECTOMY



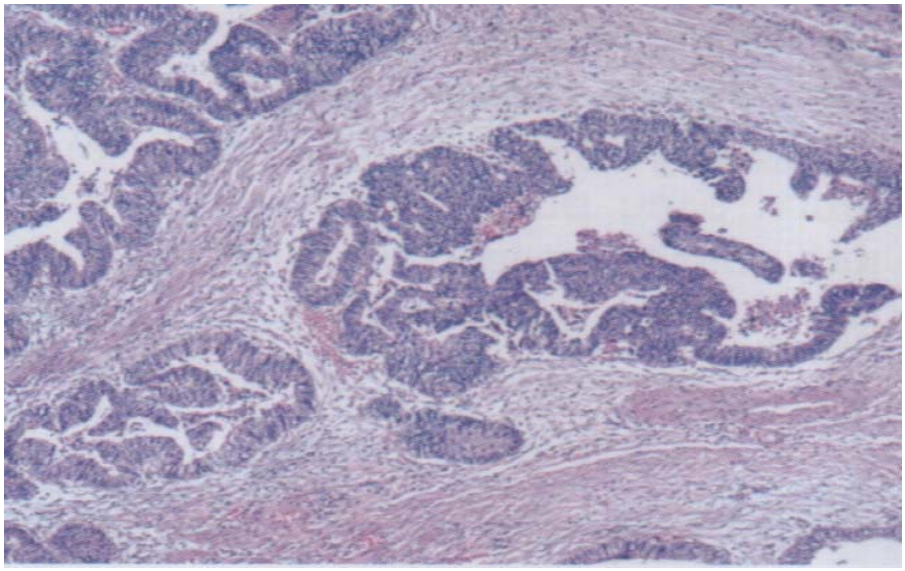
AFTER RIGHT HEMICOLECTOMY



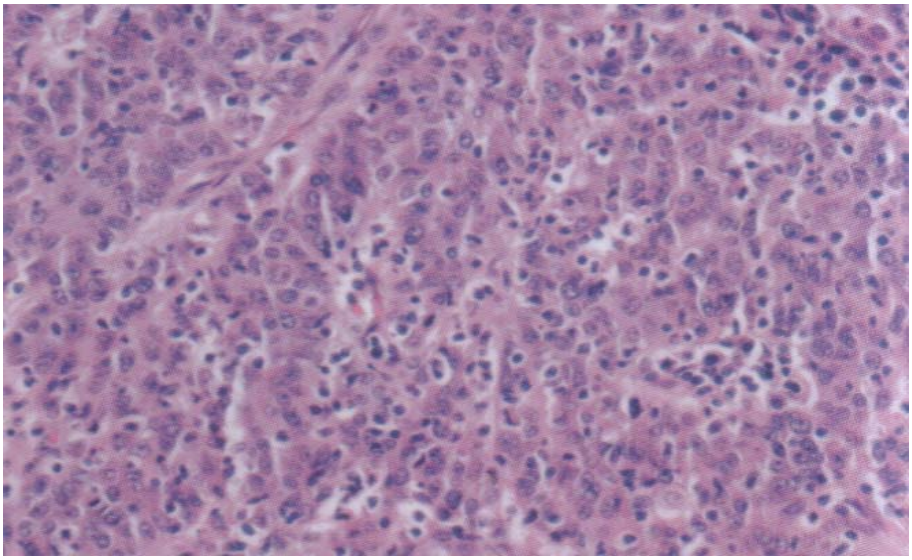
WELL DIFFRENTIATED ADENOCARCINOMA



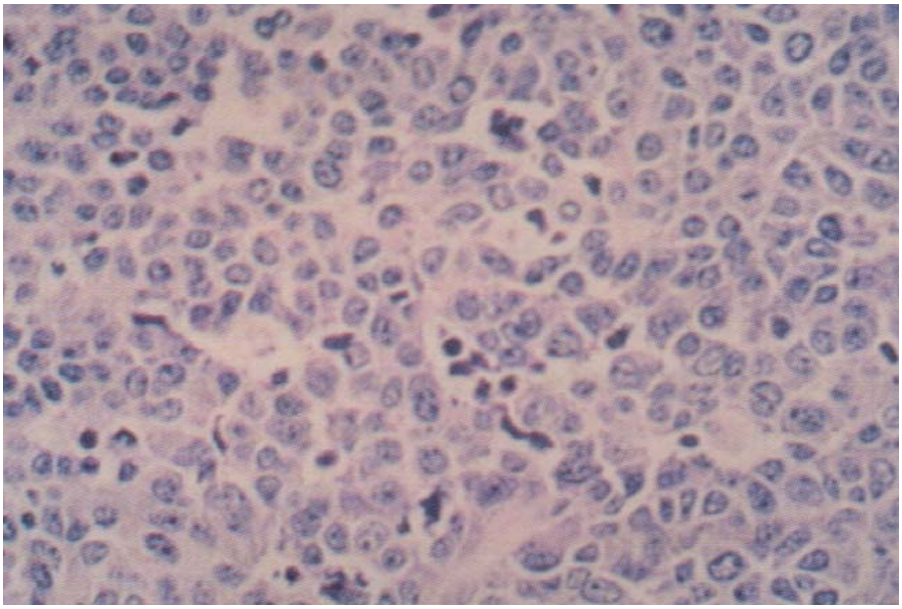
MODERATELY DIFFRENTIATED ADENOCARCINOMA



POORLY DIFFRENTIATED ADENOCARCINOMA



UN DIFFRENTIATED ADENOCARCINOMA



COLORECTAL CARCINOMA – PROFORMA

Name

Age

Sex

IPNO

WARD

Address

Socioeconomic Status

Unit

DOA

DOS

DOD

HISTORY

1. Pain Abdomen - Duration
   Nature
   Radiation
   Relation to food
   Aggravating factors
   Relieving Factors
2. Lump Abdomen - Duration
   Rate of Growth
3. Change in bowel habit - Constipation
   Diarrhoea
   Tenesmus
   Sense of uncomplete defaecation
   Mucus (or) blood in stool

4. Bleeding per rectum - Type and frequency of bleeding
Quantity and quality of blood lost
Relationship of defaecation
Mode of onset
Duration
Progress

5. Dyspepsia

6. Anaemaia

7. Ball rolling movements

8. Jaundice

9. Melaena

10. Loss of appetite

11. Loss of weight

12. Low back pain

13. Urinary symptoms

Past History

Previous Surgery
Drug Intake
Tuberculosis
Diabetes
Hypertension

Personal History

Smoker
Alcoholic

Veg/Nonveg

Food habits - animal Fat, vegetables, fruits, rice, salt

Family History

Relevant / Non Relevant

General Examination

Anaemia - P/A

Jaundice - P/A

Dehrdation - P/A

Pedal Edema - P/A

Generalised lymphadenopathy - P/A

Vital parameters

CVS

RS

Abdomen

Umbicus

VGP / VIP

Lump - Visible / Palpable

Size

Shape

Extent

Consistency

Movement with respiration

P / A - Intrinsic - P / A

Tenderness

Gaurding / Rigidity

Liver

Spleen

Free fluid

Any other mass

Hernial Orifices

P / R -

Investigations

Urine - Alb
Sugar

Blood - TC
DC
ESR
Hb%

Blood - Urea
Sugar

Serum Creatinine

LFT

X-ray, Abdomen

X-ray Chest

ECG

USG - Abdomen

Barium Enema

Proctosigmoidoscopy

Findings
Biopsy

Flexible Fibrooptic Colonoscopy

Findings
Biopsy

Faecal Occult Blood

CT Scan Abdomen

MRI scan

Others

Treatment

Surgery

Emergency/Elective

Preoperative Staging

Tumour - T1/T2/T3/T4

Nodal Status - N0/N1/N2/N3

Metastasis - M0/M1

Emergency Procedure

1) Palliative Procedures
2) Curative Procedures

Elective Procedure

1) Biopsy
2) Curative Surgery
3) Palliative Surgery

Others

Blood Transfusion - Units

Post Operative Periods

1. Wound Infection
2. Haemorrhage
3. Anastomotic Leak
4. Colostomy - Complication
5. Death - Cause
6. Others

Radiotherapy, Chemotherapy, Others

MASTER CHART

No	Name	Age/sex	I.P. NO.	Biopsy	Diagnosis	Treatment
1.	Mariammal	50/F	21229	Mod Diff Adeno Ca	CA. Rectum	Anterior resection with end to end anastomoses
2.	Sabeena beevi	50/F	21929	Mod Diff Adeno Car	CA Ascending colon	Rt Hemicolectomy
3.	Sethu	45/M	33932	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
4.	Chandra	45/F	39925	Well Diff Adeno Car	CA Splenic flexure	Lt Hemicolectomy
5.	Shenbaga rahini	40/F	44117	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
6.	Gunasingh	51/M	44992	Well Diff Adeno Car	CA Rectum	APR with permanent Colostomy
7.	Subramanian	34/M	04309	Well Diff Adeno Car	CA Rectum	APR with permanent Colostomy
8.	Murugesan	40/M	12406	Mod Diff Adeno Car	CA Splenic flexure	Lt Hemicolectomy
9.	Murugan	40/M	12476	Mod Diff Adeno Car	CA Caecum	Rt Hemicolectomy
10.	Meenakshi sundaram	80/M	14586	Poorly Diff Adeno Car	CA Splenic flexure	Lt Hemicolectomy
11.	Thirunavukkarasu	75/M	15678	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
12.	Sudalai	63/M	16872	Mod Diff Adeno Car	CA Trans Colon	Lt Hemicolectomy
13.	Kandasamy	50/M	17872	Mod Diff Adeno Car	CA Trans Colon	Lt Hemicolectomy
14.	Poomari murugaiah	34/M	18577	Well Diff Adeno Car	CA Ascending colon	Rt Hemicolectomy
15.	Rengan	25/M	21125	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
16.	Periasamy	35/F	27893	Mod Diff Adeno Car	CA Ascending colon	Rt Hemicolectomy
17.	Lakshmi	50/F	30615	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
18.	Gurusamy	58/M	31189	Mod Diff Adeno Car	CA Descending colon with int obstruction	Transverse Colostomy
19.	Murugan	54/M	33491	Mod Diff Adeno Car	CA Sigmoid colon	Resection & end to end anastomosis
20.	Sundaram	56/F	32865	Well Diff Adeno Car	CA Rectum with intestinal obstruction	Transverse Colostomy
21.	Kalathivel	51/M	31395	Mod Diff Adeno Car	CA Splenic flexure	Lt Hemicolectomy
22.	Rajeswari	38/F	36827	Poorly Diff Adeno Car	CA Rectum	APR with permanent Colostomy
23.	Saraswathy	47/F	37633	Mod Diff Adeno Car	CA hepatic flexure	Extended Rt Hemicolectomy

24.	Parameswari	27/F	50907	Mod Diff Adeno Car	CA Rectum	APR with permanent Colostomy
25.	Velammal	38/F	05678	Mod Diff Adeno Car	CA. Ascending colon	Rt. Hemicolectomy
26.	Shahul Hameed	62/M	O5912	Poorly Diff Adeno Car	CA Rectum	APR with permanent Colostomy
27.	Sudalai	63/M	07001	Poorly Diff Adeno Car	CA. Caecum	Rt. Hemicolectomy
28.	Marimuthu	40/M	07678	Mod Diff Adeno Car	CA. Ascending colon	Rt. Hemicolectomy
29.	Vilva	42/F	60243	Poorly Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
30.	Petchiammal	67/F	11003	Mod Diff Adeno Carr	CA. Rectum	APR with permanent Colostomy
31.	Chellapandy	54/M	11065	Mod Diff Adeno Car	CA. Caecum	Rt. Hemicolectomy
32.	Arulappan	57/M	11137	Poorly Diff Adeno Car	CA Transverse Colon with intestinal obstruction	Resection & end to end anastomosis
33.	Karuppaye	45/F	11411	Mod Diff Adeno Car	CA Splenic flexure	Lt Hemicolectomy
34.	Muthumariappan	51/M	20333	Mod Diff Adeno Car	CA. Rectosigmoid with int obstruction	Resection & end to end anastomosis
35.	Palaniammal	55/F	16688	Poorly Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
36.	Rajathy	58/F	18732	Mod Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
37.	Kanthaiyah	75/M	19037	Mod Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
38.	Lakshmi	48/F	21160	Mod Diff Adeno Car	CA. Rectosigmoid with int obstruction	Resection & end to end anastomosis
39.	Murugappan	52/M	21375	Mod Diff Adeno Car	CA. Caecum	Rt Hemicoloectomy
40.	Petchiammal	65/F	21552	Poorly Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
41.	Nallamuthu	80/M	23409	Mod Diff Adeno Car	CA Sigmoid Colon with int obstruction	Transverse Colestomy
42.	Selvi	31/F	24678	Mod Diff Adeno Car	CA Sigmoid Colon	Resection & end to end anastomosis
43.	Subbammal	65/F	24912	Mod Diff Adeno Car	CA Rectum	Biopsy
44.	Mary	45/F	25032	Well Diff Adeno Car	CA Caecum	Rt. Hemicolectomy

45.	Muthulakshmi	50/F	25606	Mod Diff Adeno Car	CA Sigmoid Colon	Resection & end to end anastomosis
46.	Mariammal	30/F	28114	Mod Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
47.	Kumarasamy	67/M	29067	Poorly Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
48.	Murugan	58/M	29367	Mod Diff Adeno Car	CA. Caecum	Rt hemicolectomy
49.	Sundaram	50/M	29981	Mod Diff Adeno Car	CA. Caecum	Rt hemicolectomy
50.	Ratham	70/M	30456	Mod Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
51.	Lakshmi	40/F	30567	Well Diff Adeno Car	CA. Rectum	APR with permanent Colostomy
52.	Manickam	65/M	33931	Well Diff Adeno Car	CA. Sigmoid colon with int obstruction	Resection & end to end anastomosis
53.	Subbulakshmi	65/F	34957	Well Diff Adeno Car	CA. Hepatic flexure	Rt hemicolectomy
54.	Manickam	56/M	31452	Mod Diff Adeno Carr. Diff	CA. Rectum	APR with permanent Colostomy
55	Syed Basutheen	66/M	33751	Mod Diff Adeno Carr. Diff	CA. Transverse colon	Resection and end to end anastomosis